NON- DIABETIC KIDNEY DISEASES IN PATIENTS WITH DIABETES MELLITUS (CLINICOPATHOLOGICAL CORRELATION)

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Submitted By

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DECLARATION BY THE CANDIDATE

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Dedicated

to

my Family

&

Teachers

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DR. ROHIT GHAI

LIST OF ABBREVIATIONS

DKD: Diabetic kidney disease

NDKD: Non-diabetic kidney disease

FSGN: Focal segmental glomerular nephritis

DR: Diabetic retinopathy

DM: Diabetes mellitus

DN: Diabetic nephropathy

T1DM: Type one diabetes mellitus

T2DM: Type 2 diabetes mellitus

ESKD: End stage kidney disease

CKD: Chronic kidney disease

ATN: Acute tubular necrosis

ATIN: Acute tubulointerstitial nephritis

AIN: Acute interstitial nephritis

RPGN: Rapidly proliferative glomerulonephritis

IRGN: Infection related glomerulonephritis

MGN: Membranous glomerulonephritis

GFR: Glomerular filtration rate

NPDR: Non proliferative diabetic retinopathy

AKI: Acute kidney injury

GN: Glomerulonephritis

UPCR: Urine protein creatinine ratio

ANA: Anti-nuclear antibodies

ANCA: Anti-neutrophilic cytoplasmic antibody

ABSTRACT

Introduction: Clinically indicated kidney biopsies in diabetic patients are usually performed when the presentation raises suspicion for conditions other than DKD, such as sudden onset proteinuria less than 5 years from the onset of type 1 diabetes, proteinuria in the absence of retinopathy (especially in type 1 diabetes) or neuropathy, acute kidney injury, active urinary sediment, or hematuria. Therefore, it is not surprising to find a high incidence of NDKD in clinical biopsies from diabetic patients.

Aim of Study: To study the prevalence and clinical predictors of NDKD in type 2 Diabetes Mellitus patients.

Materials & methods: This was a cross-sectional observational study that will be conducted in Dayanand Medical College and Hospital, Ludhiana. Patients suspected of NDRD in patients of diabetes underwent kidney biopsy after informed consent based on following criteria alone or in combination:

1 Hematuria (RBC >5/hpf, RBC casts)
2 Sudden increases in serum creatinine by >2 mg/dL
3 Sudden onset nephrotic syndrome
4 Renal failure in absence of diabetic retinopathy (DR)
5 Duration of DM <5 years
6 Massive proteinuria (nephrotic range) with normal renal functions
7 Significant renal insufficiency (serum creatinine >2 mg/dL) with normal or insignificant proteinuria. (<500 mg/dL)..

Results:

1. 24 out of 43 patients had pure NDKD (56.0%). 15 patients (35%) had DKD and 4 patients (9.3%) had a mixed renal disease.
2. AIN (12%) was the most common NDKD followed by IgA nephropathy, crescentic glomerulonephritis and focal proliferative glomerulonephritis (7.0% each). In mixed renal disease, the most common pathology seen was DN with AIN.
3. Hypertension duration in non-diabetic kidney disease group was 4.98 ± 2.86 years and in the DKD group it was 8.07± 4.65 years which was statistically significant.
4. Shorter duration of DM (<5 years) was seen in a greater number of patients in NDKD group when compared to the DKD group. DM duration of more than 10 years was seen in higher percentage of people in the DKD group.
5. The DKD group had more patients with NPDR as compared to the NDKD group.

Conclusion: Our study showed that early suspicion and diagnosis of NDKD is vital as many pathologies such as MN, IgA nephropathy and AIN are often treatable or even curable.

Keywords: Diabetes mellitus, non-diabetic kidney disease, Renal biopsy, nephropathy.

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INTRODUCTION

In the last few decades, diabetic nephropathy has become the leading cause of end-stage kidney disease worldwide. The increase in the number of patients with ESKD caused by diabetic nephropathy is the consequence of a constant increase in the prevalence of diabetes mellitus, especially type 2 DM, which is more frequent than type 1, as well as prolongation of the life expectancy of diabetic patients who experience such late complications.

There is a wide variation of prevalence of NDRD. [1] Type 1 diabetes mellitus is less common compared to type 2 diabetes mellitus. Non-diabetic renal disease (NDRD) occurrence ranges from 5% to 71% in different research studies. It is observed in 26.7% of Asian and 22% of European patients. Olsen, [2] in a meta-analysis, noted that the prevalence of glomerulonephritis varies from 0 to 66%, possibly due to differences in selection criteria and geographic locations. Various studies have identified older age in diabetes mellitus, the absence of neuropathy and retinopathy, and the presence of other systemic diseases as indicators of NDRD.

An estimated 537 million adults worldwide are living with diabetes, with 20%–40% possibly experiencing some level of kidney complications. This underscores the significant global impact of kidney disease among individuals with diabetes.

Other forms of kidney disease in diabetics can occur known as non-diabetic renal diseases: glomerular (membranous nephropathy), tubulointerstitial or vascular diseases. [3] Early diagnosis of non-diabetic kidney disease is vital for early treatment of patients, which can greatly slow down or completely stop the progression of chronic kidney disease to end-stage renal failure.

Many forms of non-diabetic kidney disease can be successfully treated (e.g. glomerulonephritis with immunosuppressives therapy), in contrast to diabetic nephropathy, which in the developed form with manifest proteinuria has frequently progressive course and leads to end stage renal failure in a large percentage. So, diagnosis non-diabetic kidney

disease in patients with DM is vital, as this significantly improves the prognosis of the disease.

Proteinuria in diabetic patients is usually taken as a clinical sign of diabetic nephropathy. However, not all diabetic patients with proteinuria have DN. Nondiabetic kidney disease is also seen to cause proteinuria in diabetic patients.

It remains unclear which clinical factors have greater value in the prediction of NDRD. As the reported incidence of NDRD in type 2 DM is high, it is necessary to predict, diagnose, and treat the concurrent glomerular diseases because of the prognostic and therapeutic importance. The following factors influence the development of NDKD.

1. Hypertension: High blood pressure is a leading cause of kidney damage. Chronic hypertension can lead to glomerulosclerosis, characterized by scarring of the kidney tissue.
2. Glomerular Diseases: Conditions such as glomerulonephritis, an inflammatory condition of the glomeruli, are common and can be primary or secondary to infections or autoimmune diseases.
3. Chronic Interstitial Nephritis: Often due to long-term use of certain medications, exposure to toxins, or chronic infections.
4. Genetic Disorders: Polycystic kidney disease (PKD) and other genetic conditions can lead to CKD.
5. Obstructive Uropathies: Conditions such as kidney stones, tumours, or congenital abnormalities that obstruct urine flow can cause chronic kidney damage.
6. Infectious Diseases: Tuberculosis, malaria, and other endemic infectious diseases can lead to CKD.

Socioeconomic and Environmental Factors

The living conditions, access to healthcare, and education levels in various parts of India significantly influence the incidence and management of NDKD. Rural areas often face greater challenges such as:

1. Limited Access to Healthcare: Delay in diagnosis and treatment due to inadequate healthcare facilities.

2. Diet and Lifestyle: Malnutrition, poor dietary habits, and lack of awareness about preventive measures also contribute to NDKD.

Diagnosis and Management

Early detection is crucial for effective management of NDKD. Regular screening for kidney function through blood tests (serum creatinine) and urine tests (for proteinuria) is vital. Imaging studies like ultrasound can also help in the diagnosis.

Kidney biopsy is the tool of choice for diagnosing non-diabetic renal disease in diabetic patients. It is a guideline for treatment. Kidney biopsy can find out that a patient has diabetic nephropathy, cohabitating diabetic nephropathy and other non-diabetic renal disease or only non-diabetic renal disease.

KDOQI Clinical Practice Guidelines[4] from 2007 suggest clinical features that can be indicative of the presence of non-diabetic renal disease in diabetic patients: absence of diabetic retinopathy, rapidly deteriorating renal function, proteinuria of increasing severity, or nephrotic syndrome development, refractory hypertension, active urinary sediment (microscopic hematuria with red blood cell or granular casts), signs or symptoms of systemic disease or GFR decline of > 30% within two to three months

Non-diabetic kidney disease is a complex and multifaceted issue in India, requiring a multi-pronged approach for effective management and prevention. Enhanced awareness, early detection, robust healthcare infrastructure, and continuous research are essential components to address the burden of NDKD in the country.

Thus, we conducted the study non-diabetic kidney disease in patients with diabetes mellitus (clinic-pathological correlation).

AIMS AND OBJECTIVES

To study the prevalence and clinical predictors of NDKD in type 2 Diabetes Mellitus patients.

REVIEW OF LITERATURE

Non-diabetic kidney disease is a significant health concern in India, affecting a large portion of the population. Unlike diabetic kidney disease, which is commonly linked to the broad prevalence of diabetes, NDKD encompasses a variety of conditions that affect the kidneys without being directly related to diabetes.

The clinical course of non-diabetic renal disease is atypical, with the absence of changes in the target organs caused by long-term diabetes (retinopathy), usually more abundant proteinuria that occurs after a shorter duration of diabetes than in diabetic nephropathy, and the presence of dysmorphic erythrocytes and erythrocyte casts in urine indicating glomerulonephritis. Examples of NDKD include ATN, AIN, RPGN, IgA nephropathy, IRGN etc

Diabetic kidney disease is a clinical syndrome in a patient with diabetes mellitus characterized by persistent albuminuria (>300 mg/day) on at least two occasions 3-6 months apart, elevated blood pressure and progressive decline in renal function. [5] DKD is the commonest cause of chronic kidney disease and end-stage kidney disease worldwide. Around 20%-40% of patients with DM develop DKD.

Diabetic kidney disease is one of the most significant micro vascular complications of diabetes. However, in diabetic patient's kidney diseases other than DKD can develop. NDKD can occur either alone or superimposed on DKD.

Epidemiology

537 million adults globally are living with diabetes of which 20%–40% have some form of renal involvement, stressing a huge world burden of kidney disease in patients with diabetes. [6] Renal issues in individuals with type 2 diabetes may sometimes be attributed to factors unrelated to diabetes, commonly known as non-diabetic kidney disease. In addition, some patients may have renal involvement due to other etiologies other than diabetes (mixed kidney disease group, having features of both DKD and NDKD).

The treatment of DKD involves tight blood pressure and glycaemic control. However, in a significant proportion of NDKD, there are certain specific treatment options including steroids and other immunosuppressants which could considerably improve patient outcomes. In addition, appropriate diagnosis could help in prognostication. [6]

Histopathology is the gold standard to tell apart DKD from NDKD. Studies suggest that subjects with diabetes have fluctuating proportion of DKD and NDKD. This difference in prevalence could be because of differences in clinical practice where criteria followed by clinicians to subject a patient to renal biopsy.

As renal biopsy is invasive with limited obtainability of expertise, widespread use of renal biopsy is difficult in such scenarios.

Several clinical parameters have been used to help recognize individuals at greater risk of NDKD. Unfortunately, data are scant to support the use of the same. Results from retrospective meta-analysis of several studies have suggested certain clinical criteria to help identify patients with greater possibility of NDKD.

Types of non-diabetic kidney disease

1. IgA nephropathy

Most frequently observed in children and young adults, immunoglobulin a nephropathy is identified by the prevalent deposition of IgA in the glomerular mesangium. It stands as a major cause of glomerulonephritis. Conditions like Henoch-Schönlein purpura (HSP) have a strong connection to IgA nephropathy, while other systemic diseases, such as ankylosing spondylitis, commonly exhibit IgA deposits in the mesangium.

2. Crescentic glomerulonephritis

Rapid decrease in the glomerular filtration rate (GFR) of at least 50% over a short period, from a few days to 3 months. Pathologic finding is extensive glomerular crescent formation and focal rupture of glomerular capillary walls that can be seen by light microscopy and electron microscopy.

3. Focal proliferative glomerulonephritis

Focal proliferative glomerulonephritis falls under a pathologic category commonly found in IgA nephropathy and lupus nephritis. This type of glomerulonephritis is identified in renal biopsy specimens as class III IgA nephropathy in Haas classifications.

Definition: 50% or less of glomeruli are hypercellular; global hypercellularity may be present but no more than 50% of glomeruli are hypercellular. Commonly seen in IgA nephropathy, lupus nephritis and good pastures syndrome

4. Membranous nephropathy

Membranous nephropathy is the most common cause of nephrotic syndrome in the adult population, but also occurs in children. Approximately 80% of MGN cases are idiopathic and the remainder are secondary to malignancy, infectious disease, or an autoimmune disorder.

Immune complexes deposit along the subepithelial region of the glomerular basement membrane.

5. Post infectious glomerulonephritis

Postinfectious glomerulonephritis, a type of nephritic syndrome, is the primary glomerular disorder affecting children aged between 5 and 15 years. [7] It is uncommon in children under 2 years old and adults over 40 years old. The majority of cases are linked to nephritogenic strains of group A beta-haemolytic streptococci. Other less common causative agents include non-streptococcal bacteria, viruses, parasites, rickettsiae, and fungi.

Microbial antigens attach to the glomerular basement membrane, triggering the alternate complement pathway directly or through interaction with antibodies in the bloodstream. This process leads to glomerular damage, which can be either focal or diffuse.

6. Focal segmental glomerulosclerosis

Histologically characterized by segmental scarring that involves a part of the glomerulus and only affects some but not all glomeruli sampled. [8]

Patients with this condition may show symptoms of nephrotic and nephritic syndrome or renal insufficiency. The primary clinical presentation of FSGS, seen in more than 70% of patients, is nephrotic syndrome, characterized by generalized or dependent edema, fatigue, and loss of appetite. Another prevalent symptom is hypertension.

Aetiology:

a) FSGS with hyalinosis

b) Progression from immunoglobulin M (IgM) nephropathy

c) Progression from minimal-change disease

d) Progression from mesangial proliferative glomerulonephritis

e) Superimposed on other primary glomerulonephritis conditions

7. Acute tubule-interstitial Nephritis

Tubulointerstitial nephritis comprises an immune-mediated inflammatory procedure affecting the interstitium and renal tubules. The inflammation triggers the accumulation of inflammatory cells, fluid, and extracellular matrix around the interstitium, accompanied by the invasion of tubular cells.

Etiology:

- The primary cause of acute interstitial nephritis (70% to 75%) is drugs, also known as allergic interstitial nephritis.
- 20% to 25% of tubulointerstitial nephritides can be attributed to the following causes:
  i. Infections (viral, bacterial, parasitic, or fungal).
  ii. Linked to various systemic inflammatory conditions like IBD, sarcoidosis, SLE, and Sjögren's disease.

8. Acute tubular necrosis

The primary cause of acute kidney injury is acute tubular necrosis, which is inaccurately named as true cellular necrosis is typically limited. This condition affects more than just tubular structures and is linked to high rates of morbidity and mortality. The injury results in damage and death of renal tubular cells, triggered by intrarenal vasoconstriction from ischemic events, nephrotoxic effects, or a combination of both mechanisms.

Ischemic-Induced Acute Tubular Necrosis

Prerenal kidney failure and ischemic acute tubular necrosis share a similar range of causes. Diseases that result in prerenal azotemia can also lead to ischemic acute tubular necrosis. Causes include hypovolemic conditions like diarrhea, vomiting, bleeding, dehydration, burns, renal losses from diuretics, and third space sequestration. Conditions causing edema such as heart failure and cirrhosis can reduce kidney perfusion. Sepsis or anaphylaxis can lead to systemic vasodilation, while coagulopathy like disseminated intravascular coagulation can also trigger acute tubular necrosis.

Nephrotoxic-Induced Acute Tubular Necrosis

The kidney clears and processes many drugs. These drugs act as exogenous toxins and can cause direct renal tubular injury or crystal-induced acute kidney injury, leading to acute tubular necrosis. Examples are aminoglycosides, amphotericin B, radiocontrast media, sulfa drugs, and acyclovir. Intravenous immunoglobulin containing sucrose can also cause acute tubular necrosis.

Proteins such as hemoglobin and myoglobin can act as endotoxins by 3 mechanisms:

1. Causing proximal tubular injury, tubular obstruction, or renal vasoconstriction.
2. Crystal-induced nephropathy in the setting of ongoing malignancy treatment.
3. Light chain accumulation in multiple myeloma is toxic to the renal tubules.

Sepsis-Induced Acute Tubular Necrosis

Sepsis contributes to acute tubular necrosis through systemic hypotension and renal hypoperfusion. Additional mechanisms involve endotoxemia inducing kidney injury via renal vasoconstriction, the release of inflammatory cytokines prompting increased secretion of reactive oxygen species, ultimately resulting in renal damage. [9]

9. Renal amyloidosis

Proteins gather and accumulate outside cells in tissues as insoluble fibrils, leading to organ malfunction. Systemic amyloidosis typically

affects the kidneys, displaying non-cellular pathological Congo red-positive amyloid fibril deposits in glomeruli, vessels, or interstitium.

The kidney is a frequent location for amyloid buildup in light chain, serum amyloid A, and various other types of amyloidosis. The primary clinical signs of renal amyloidosis often include proteinuria, leading to nephrotic syndrome, and a gradual decline in kidney function.

Treatment for renal amyloidosis depends on the type of amyloid present and typically involves a combination of therapies aimed at addressing the root cause of the amyloidosis and providing supportive care.

Patients who progress to end-stage renal disease should be considered for either dialysis or renal transplantation.

10. Lupus nephritis

SLE, an autoimmune disease, leads to persistent inflammation and organ damage. Typically, it develops three years after the onset of SLE. Regular kidney function assessments are crucial. Even if patients do not show clinical signs of renal disease, there may still be histological proof of lupus nephritis. [10]

Aetiology

Lupus nephritis results from a type-3 hypersensitivity reaction where Anti-dsDNA attaches to DNA, creating an immune complex that settles near the kidney's glomerular basement membrane. This triggers the complement pathway, leading to the infiltration of neutrophils and other inflammatory cells.

There are genetic factors that can increase the likelihood of an SLE patient developing lupus nephritis. Variations in the allele responsible for immunoglobulin receptors on macrophages and specific APOL1 gene mutations, primarily present in African American SLE patients, have been linked to a higher risk of developing lupus nephritis.

Lupus Nephritis categorization revised by the International Society of Nephrology and the Renal Pathology Society in 2003. [11]

i. Minimal mesangial lupus nephritis

ii. Mesangial proliferative lupus nephritis

iii. Focal lupus nephritis

iv. Diffuse lupus nephritis

v. Membranous lupus nephritis

vi. Advanced sclerosis lupus nephritis

11. Acute pyelonephritis

A bacterial infection that results in kidney inflammation typically arises as a complication of a urinary tract infection those progresses from the bladder to the kidneys.

Pathophysiology

E. coli possesses a distinct capability to stick to and establish colonies in the urinary tract and kidneys, making it the predominant pathogen. The interaction of E. coli with receptors on the surface of uroepithelial cells is crucial. Neutrophils play a vital role in managing bacterial movement upward, while macrophages handle the inflammatory process and scarring response.

An acute response ensues by releasing chemokines and other inflammatory factors and this can ultimately result in scarring of the renal parenchyma.

Connective tissue replaces functional renal tissue to limit the spread of bacteria. Renal scarring occurs due to broken renal cell barriers, localized inflammation, cytokines, clotting, and hypoxia. The above mechanisms may result in complete renal involvement and may progress to sepsis and septic shock.

Histopathology

i. Sub mucosal clusters of inflammatory cells and swollen peritubular capillaries.

ii. Superficial erosions progress to frank ulceration. Acute findings include submucosal collagen, engorged peritubular capillaries, and infiltration of neutrophils, macrophages, and plasma cells in renal tissues. Additionally, plugs might develop in renal tubules and extend into the interstitium.

iii. Degeneration or damage to the renal tubules results in the formation of localized areas of necrosis and micro-abscesses in the renal parenchyma.

Signs and symptoms of non-diabetic kidney disease

1. Nephrotic rage proteinuria
2. Weight gain
3. Swelling of the ankles and legs
4. Nausea
5. Weakness
6. Anaemia
7. High blood pressure
8. Gross haematuria

Predictive factors for NDKD

1. Old age
2. Microhematuria
3. Absence of diabetic retinopathy
4. Shorter duration of diabetes mellitus
5. Anemia
6. Normal blood pressure
7. Normal HBA1C
8. Higher level of proteinuria

Pathophysiology

I. Anemia

Renal tubulointerstitial injuries are reportedly associated with anemia. The process includes erythropoietin-producing fibroblasts transforming into myofibroblasts in fibrotic kidneys. Additionally, research indicates that interstitial damage and a relative deficiency in erythropoietin leading to anemia occur during the initial phase of DN.DM causes severe tubular atrophy and interstitial scarring resulting in impaired erythropoietin production leading to anaemia. In addition, anaemia can aggravate renal fibrosis by causing renal tissue hypoxia via the stimulation of cytokine production, including hypoxia-inducing factor (HIF)-

II. Old Age

The natural aging process in the kidney presents similar histopathologic characteristics such as sclerosis, mesangial matrix expansion, thickening of tubular and glomerular basement membranes, as well as interstitial atrophy and fibrosis. These findings are comparable to those observed in DKD, indicating a common pathophysiology.

Accelerated ageing in tubular cells and podocytes has been shown in kidney biopsies from patients with diabetic nephropathy and type II diabetes mellitus. Ageing cells secrete proinflammatory cytokines and complement component and pro-fibrotic factors that push glomerulosclerosis, podocyte hypertrophy, mesangial changes, and tubulointerstitial fibrosis observed in DKD. Advanced glycosylation end products and cell surface receptor of AGEs accumulate in both diabetic and senescent kidneys.

III. Absence of diabetic retinopathy

Diabetic retinopathy and diabetic kidney disease (DKD) share similar underlying mechanisms and are often coexisting conditions.[12] According to El-Asrar et al. [13] individuals with type 1 diabetes mellitus and diabetic retinopathy (DR) have a 13-fold higher risk of developing DKD compared to those without DR. A meta-analysis revealed that patients with DR are almost four times more likely to develop DKD, while those with DKD have double the chances of being diagnosed with DR. Recent studies show an increasing trend of inconsistency between DKD and DR. In an Italian study, the disparity between DR and DKD was reported at 36.6%, reflecting a similar trend in our own research.

A study done by Liu Z et al [12] elder age, male gender, a longer duration of disease, and uncontrolled blood glucose were correlated with both DKD and DR. Numerous studies show that DKD and DR may differ. A clear percentage of patients with type 2 diabetes mellitus had DKD or DR alone. A new classification of diabetes is proposed by a Swedish group. First classification describes patients with severe insulin resistance leading to a higher incidence of kidney disease and cardiovascular disease, while the second classification, characterized

by severe insulin deficiency, was associated with a higher incidence of diabetic retinopathy. More importantly, SGLT2 inhibitors and GLP1-RA exerted exceptional renal but not retinal protective effects.

IV. Hematuria

The occurrence of microscopic hematuria in diabetic patients with proteinuria can vary from 11% to 76–78%, depending on the specific population studied and the definition of hematuria used. Microscopic hematuria is often seen as an indicator of non-diabetic renal disease (NDRD) and might prompt a biopsy in diabetic patients with proteinuria. However, some research indicates that the prevalence of hematuria in diabetic patients with histological evidence of NDRD is only slightly increased or not increased at all compared to those with biopsy-confirmed isolated diabetic nephropathy (DN). Additionally, analyzing erythrocyte morphology through phase-contrast microscopy is crucial for diagnosing the underlying causes of hematuria. Studies suggest that hematuria with dysmorphic red blood cells (RBCs) in patients diagnosed with DN could signal the presence of another type of glomerulonephritis.

V. Shorter duration of diabetes mellitus

Diabetic Kidney Disease (DKD) affects approximately 30-40% of patients who have had diabetes for more than 10 years. [14] Progression in these patients typically starts with hyperfiltration, leading to an increase in estimated glomerular filtration rate (eGFR), followed by microalbuminuria, macroalbuminuria, overt nephropathy, and eventually kidney failure over several years. Histological characteristics of DKD involve thickening of the glomerular capillary wall, mesangial expansion, Kimmelstiel-Wilson lesions, diffuse glomerulosclerosis, hyaline arteriosclerosis, and exudative lesions such as fibrin caps, capsular drops, and hyaline thrombi. Current treatments focus on slowing down the disease progression by closely managing blood sugar levels and blood pressure. Medications targeting the renin-angiotensin-aldosterone system (RAAS) and SGLT1 receptors play a crucial role in this management. Differentiating Nondiabetic Kidney

Disease (NDKD) in patients with diabetes of over 12 years is challenging. While some studies suggest microhematuria as a marker for NDKD, 33-50% of DKD patients may also present with microhematuria. The sudden onset of nephrotic range proteinuria may indicate an underlying podocytopathy. A diabetic patient showing an annual eGFR decline exceeding 10 ml/min/1.73 m^2 should raise concerns about NDKD.

Diagnosis of NDKD

Kidney biopsy is considered the gold standard for diagnosing non-diabetic renal disease in diabetic patients. It serves as a crucial tool for guiding treatment decisions and predicting outcomes. Through kidney biopsy, healthcare providers can determine if a patient has:

- Diabetic nephropathy
- Both diabetic nephropathy and another non-diabetic renal disease (typically glomerulonephritis)
- Solely non-diabetic renal disease.

Indications for renal biopsy in patients with diabetes are [3]

❖ Nephrotic syndrome in patients who had diabetes for less than 5 years or without accompanying diabetic retinopathy, or with sudden worsening of proteinuria.

* Microscopic or gross haematuria in patients with diabetes.

* Acute renal failure

* Clinically unclear sudden deterioration of chronic kidney failure in patients with diabetes.

The KDOQI Clinical Practice Guidelines from 2007[4] outline clinical features that may indicate the presence of non-diabetic renal disease in diabetic patients. These include:

- Absence of diabetic retinopathy
- Rapidly deteriorating renal function
- Increasing severity of proteinuria or development of nephrotic syndrome

- Refractory hypertension
- Active urinary sediment (microscopic haematuria with red blood cell or granular casts)
- Signs or symptoms of systemic disease
- GFR decline of more than 30% within two to three months.

MATERIAL AND METHODS

A cross-sectional observational study that was conducted in Dayanand Medical College and Hospital, Ludhiana. Patients suspected of NDRD in patients of diabetes underwent kidney biopsy after informed consent. NDRD will be suspected in patients of diabetes mellitus when they presented with any of the following:

1. Hematuria (RBC >5/hpf, RBC casts)
2. Sudden increases in serum creatinine by >2 mg/dL
3. Sudden onset nephrotic syndrome
4. Renal failure in absence of diabetic retinopathy (DR)
5. Duration of DM <5 years
6. Massive proteinuria (nephrotic range) with normal renal functions
7. Significant renal insufficiency (serum creatinine >2 mg/dL) with normal or insignificant proteinuria. (<500 mg/dL).

These patients were be subjected to kidney biopsy to confirm the presence of NDRD.

Inclusion criteria: Patients suspected of NDKD in patients of type 2 diabetes mellitus who underwent kidney biopsy.

Exclusion criteria:

Patient were excluded if they meet any of the following criteria:

1. Ongoing urinary tract infection
2. Calculus renal disease
3. Obstructive uropathy
4. Renal tumors
5. Bleeding diathesis
6. Bilateral contracted kidneys (kidney size no less than 8cm)
7. Single contracted kidney (contraindication for biopsy)
8. Patients not consenting for kidney biopsy
9. Patients who don’t give consent for study

Clinical Significance

Diabetic nephropathy (DN) is the leading cause of end stage renal disease worldwide. Despite this, most patients with diabetes mellitus (DM) are not formally evaluated with a renal biopsy. Kidney biopsy can differentiate DN from non-diabetic renal diseases (NDRD), but it is invasive, not suitable for every patient, associated with serious complications (though rare) and other issues like availability and reluctance, especially more in developing countries like India. The diagnosis is mostly made on clinical grounds. Proteinuria or renal failure in diabetic patients is usually interpreted as a clinical manifestation of DN. However, not all diabetic subjects with proteinuria or renal failure have DN. Non-diabetic renal diseases, though rarer than DN, have been seen to cause proteinuria or renal failure in diabetics. The occurrence of NDRD in type 1 diabetes mellitus (T1DM) is rare compared with those with type 2 diabetes mellitus (T2DM). It has been well demonstrated that renal disease in patients suffering from T1DM for over 10 years is usually the result of DN, as proven histologically in >95% of these patients. It is usually believed that DN is hard to reverse and further management aims at prevention of progression of disease. But some NDRDs such as minimal change disease, immunoglobulin A (IgA) nephropathy and membranous nephropathy are often treatable and even curable. The therapy and prognosis of DN and NDRD are quite different and the differential diagnosis is of considerable importance. Hence, it is necessary to suspect, diagnose and treat the concurrent glomerular diseases or the unrelated renal disorders that are superimposed on DN because of the prognostic and therapeutic implications.

Keywords: Diabetes mellitus, non-diabetic kidney disease, Renal biopsy, diabetic nephropathy.

OBSERVATION AND RESULTS

Table 1: Age distribution (n=43)

Age group	No. of cases	Percentage
< 40	4	9.3%
41-50	8	18.6%
51-60	13	30.2%
61-70	12	27.9%
> 70	6	14.0%
Total	43	100.0%
Mean +/- sd	57.16 +/- 11.34	
Min-max.	31-76.0	

The majority of patients, i.e. 13 patients of the total 43 patients were in the age group 51-60 years (30.2%), followed by 12 patients in the group 61-70 years (27.9%), 8 patients fell in the 41-50 years age group (18.6%), 6 patients were greater than the age of 70 (14%) and 4 patients were less than 40 years (9.3%).

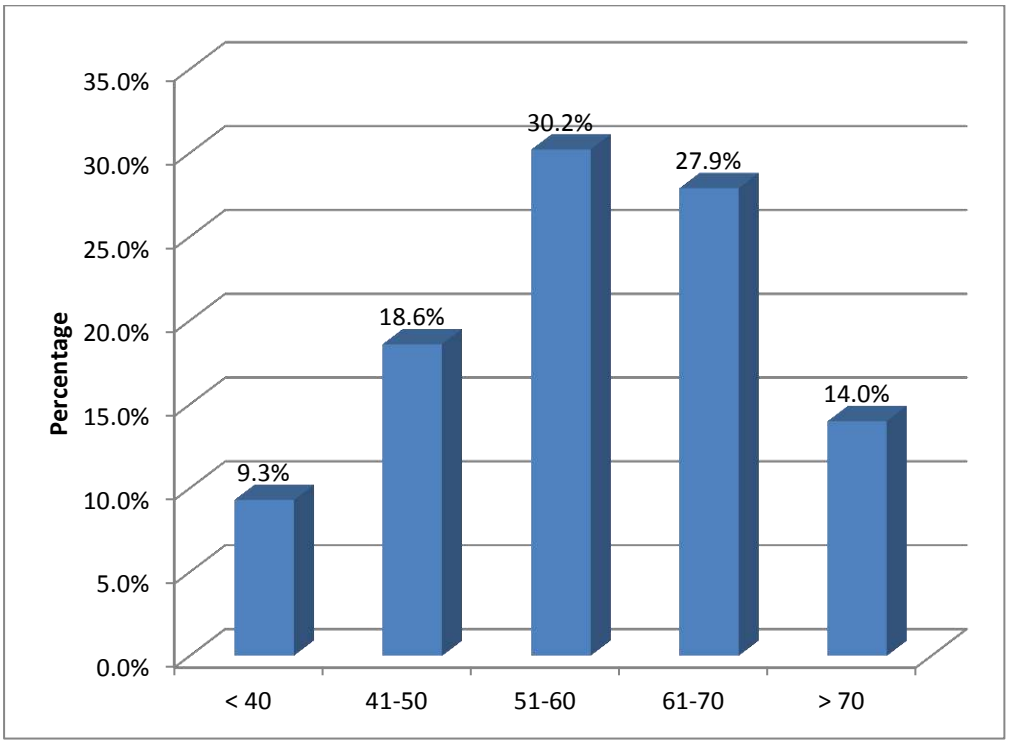

Figure 1: Age distribution (n=43)

Table 2: Sex distribution (n=43)

Sex	No. of cases	Percentage
Females	16	37.2%
Males	27	62.8%
Total	43	100.0%

27 out of the 43 patients were males (62.8%) and 16 patients were females (37.2%). The male to female ratio was 1:1.7

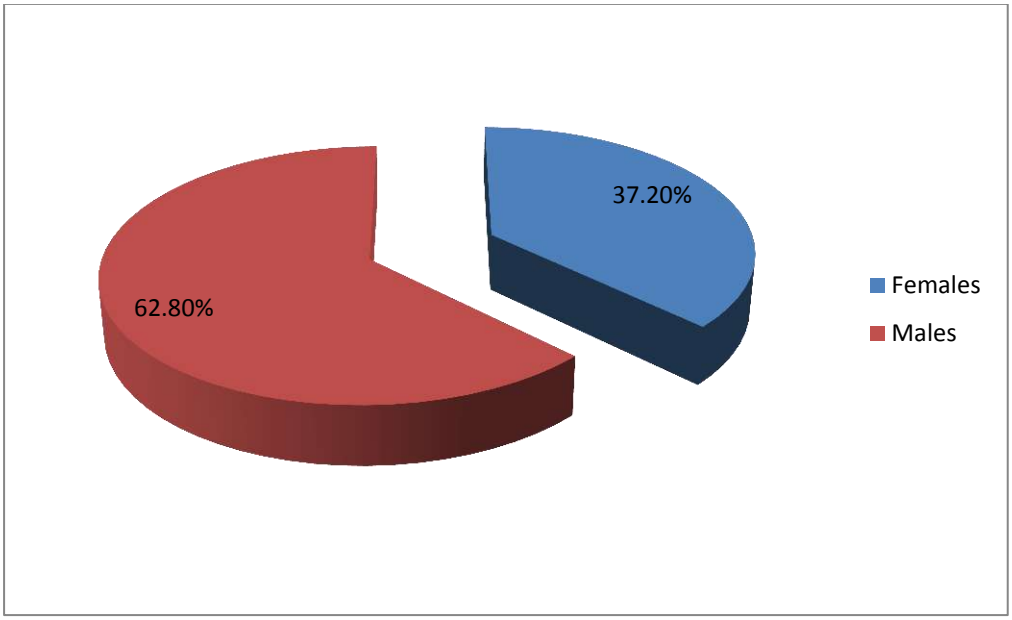

Figure 2: Sex distribution (n=43)

Table 3: Presenting symptoms (n=43)

History of present illness	No. of cases	Percentage
Dyspnoea	16	37.2%
Chest pain	0	0.0%
Oliguria	17	39.5%
Facial puffiness	12	27.9%
Oedema feet	28	65.1%
Haematuria	2	4.7%

28 out of 43 had edema feet (65.1%), followed by 17 patients having oliguria (39.5%), 16 patients had dyspnea (37.2%), 12 patients had facial puffiness (27.9%), 2 patients had hematuria (4.7%) and none of the patients had chest pain.

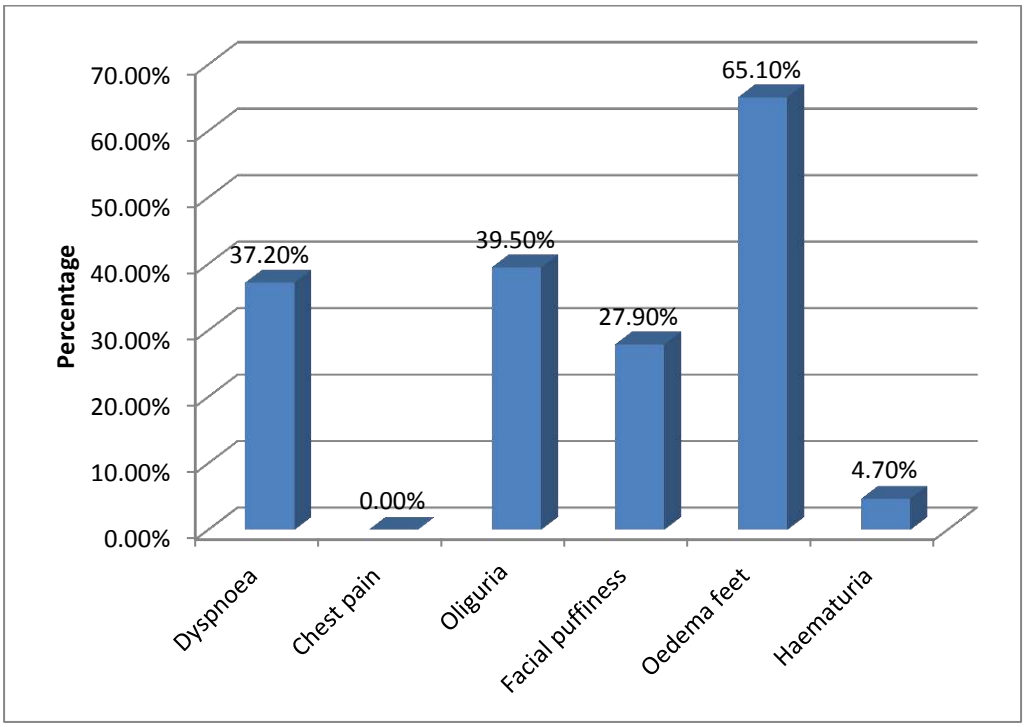

Figure 3: Presenting symptoms (n=43)

Table 4: Personal history (n=43)

Addiction	No. of cases	Percentage
Smoking	10	23.0%
Alcohol	9	21.0%
Other substance use	0	0.0%

10 patients were smokers, and 9 patients consumed alcohol. Smokers and alcohol consumers were only males.

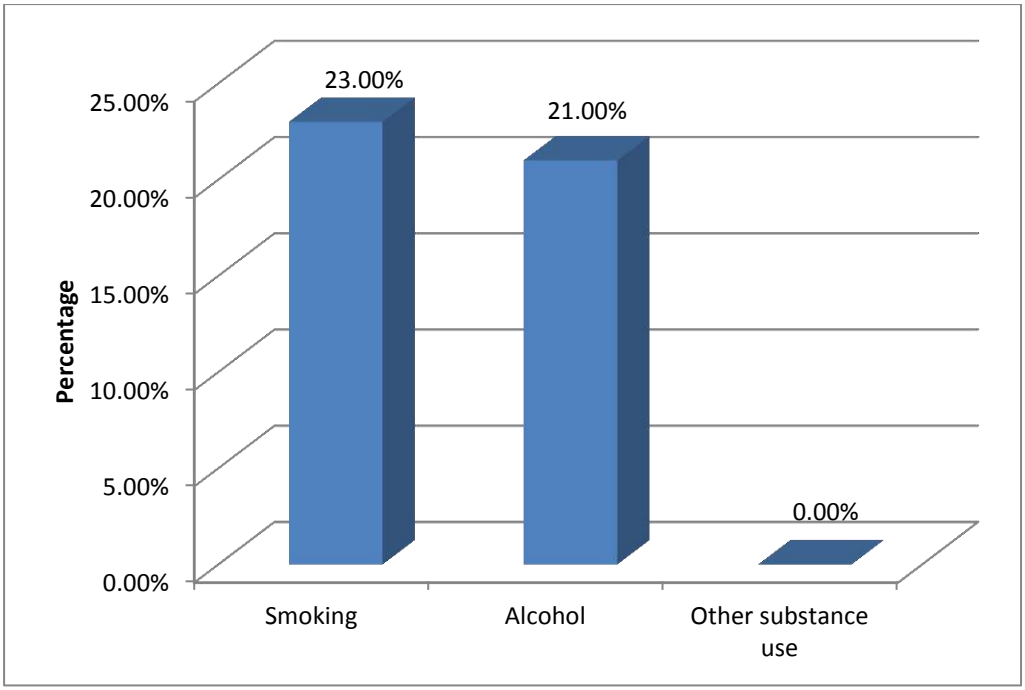

Figure 4: Personal history (n=43)

Table 5: Drug intake (n=43)

Drug intake (NSAID's, antibiotics, indigenous medications, etc.)	No. of cases	Percentage
NSAID's	6	14.0%
Indigenous medicine	2	4.7%
No	35	81.4%

6 patients were taking NSAID's regularly (14%) and 2 patients were taking alternative forms of treatment (4.7%).

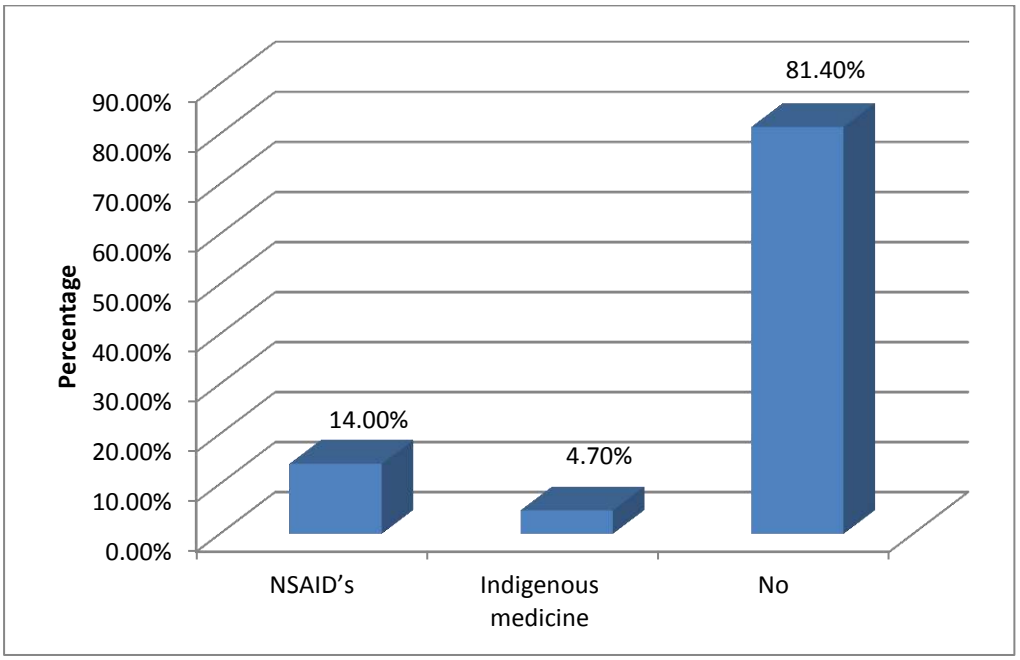

Figure 5: Drug intake (n=43)

Table 6: General physical examination (n=43)

	No. of cases	Percentage
Oedema feet	28	65.1%
Pallor	8	18.6%
Icterus	0	0.0%
Clubbing	0	0.0%
Cyanosis	0	0.0%
JVP	0	0.0%

28 patients out of 43 (65.1%) had pedal edema on physical exam and pallor was noted in 8 patients (18.6%). None of the patients had raised JVP, cyanosis, clubbing or icterus.

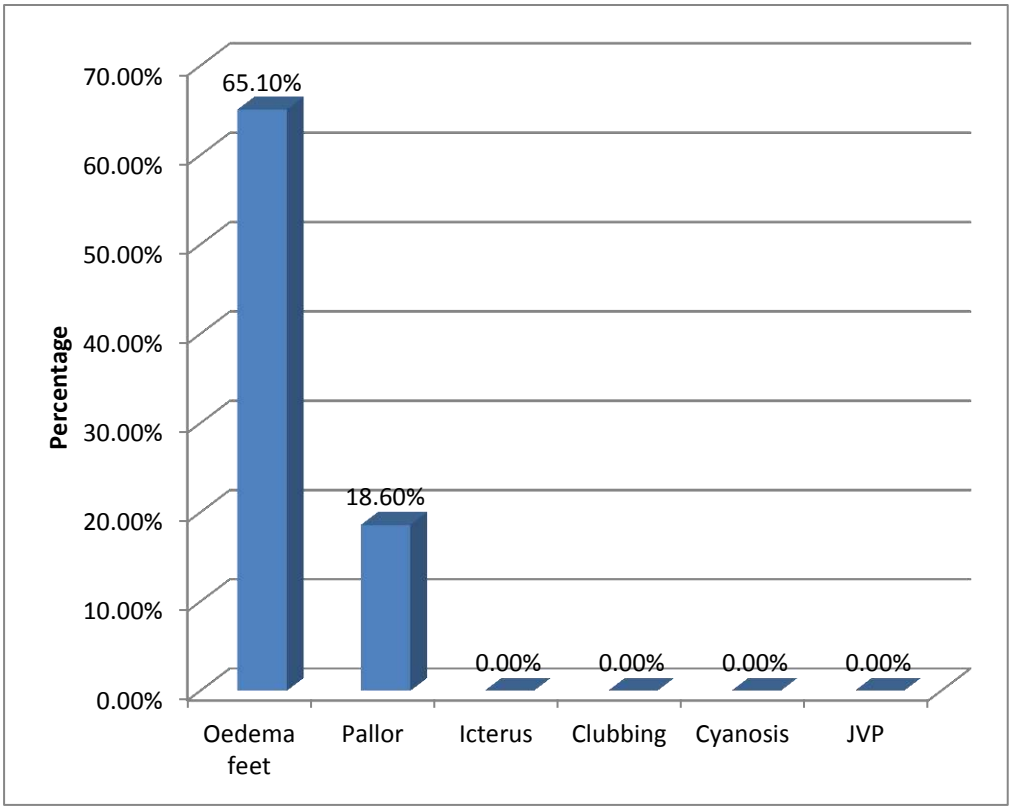

Figure 6: General physical examination (n=43)

Table 7: Respiratory examination (n=43)

Respiratory exam:	No. of cases	Percentage
Added sounds	9	20.9%
Normal respiratory exam	34	79.1%
Total	43	100.0%

Majority of patients had a normal respiratory exam (79.1%) while 9 patients (20.9%) had added sounds in the form of rhonchi and crepitations.

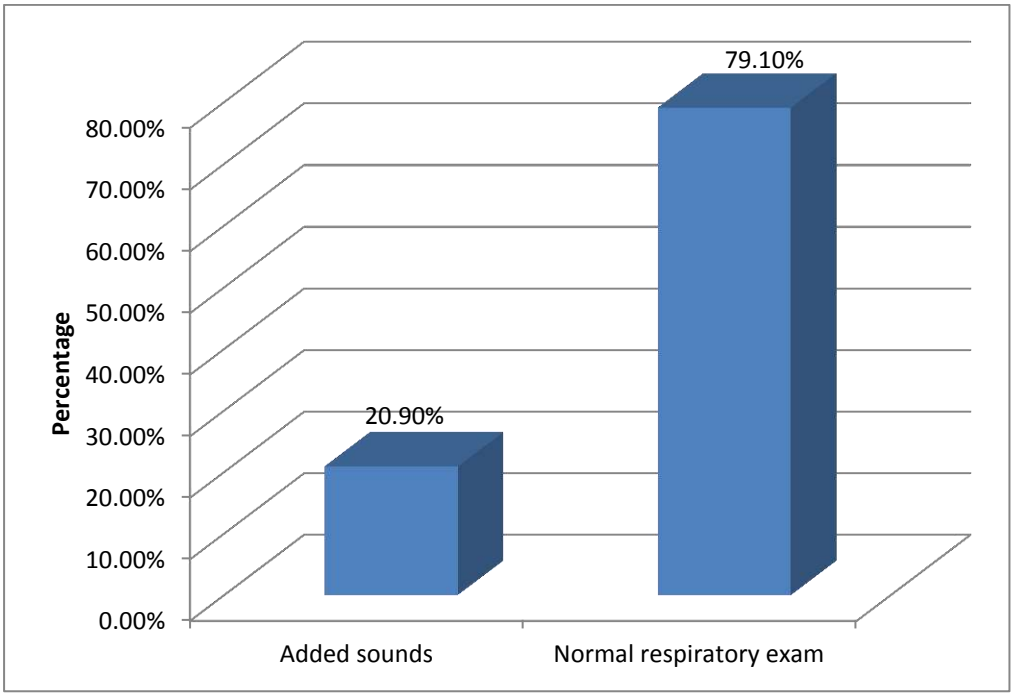

Figure 7: Respiratory examination (n=43)

Table 8A: Hypertension Duration (n=43)

Hypertension years	No. of cases	Percentage
No HTN	9	20.9%
5 to 10	4	9.3%
> 10	30	69.8%
Total	43	100.0%

Table 8B: Hypertension on regular treatment (n=43)

Hypertension on regular treatment	No. of cases	Percentage
No	14	41.1%
Yes	20	58.8 %
Total	34	100.0%

34 out of 43 patients were hypertensive out of which 30 patients were hypertensive for more that 10 years. 9 patients were non-hypertensive. 20 patients (58.8%) were on regular treatment and 41% patients were non-compliant for the same.

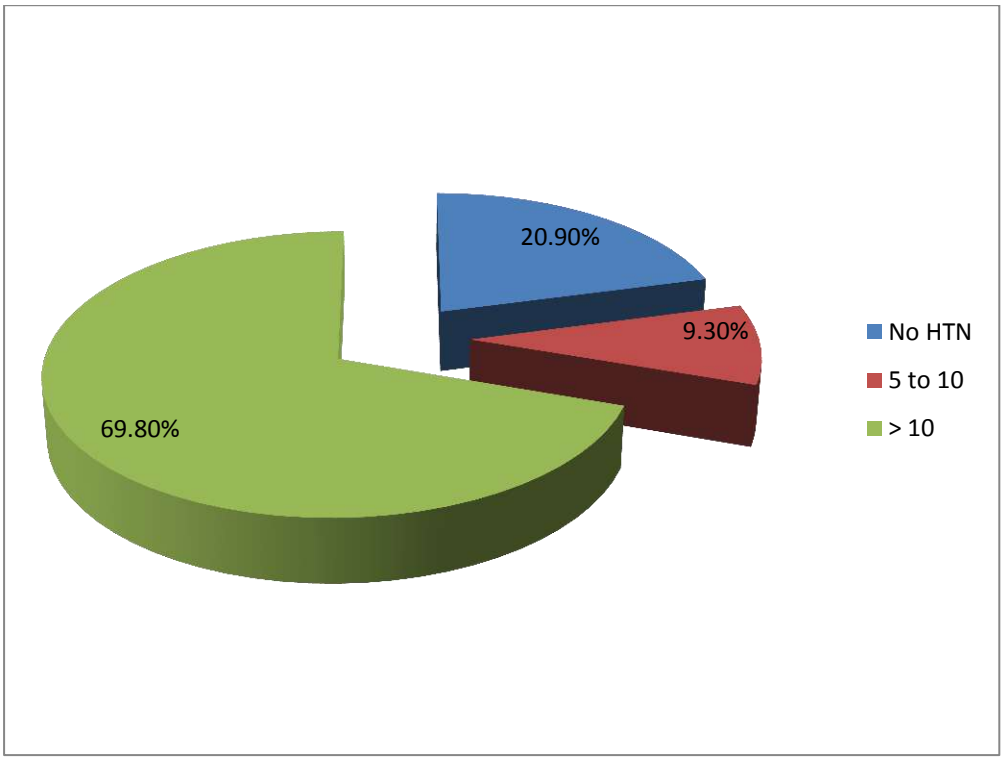

Figure 8A: Hypertension Duration (n=43)

Table 9: No. of Diabetes mellitus patients on regular treatment (n=43)

Diabetes Mellitus	Patients	Percentage %
On regular treatment	33	74%
Non-compliant	11	26%
Total	43	100%

11 patients were non-compliant with their diabetes medications. Majority of patients were on regular treatment.

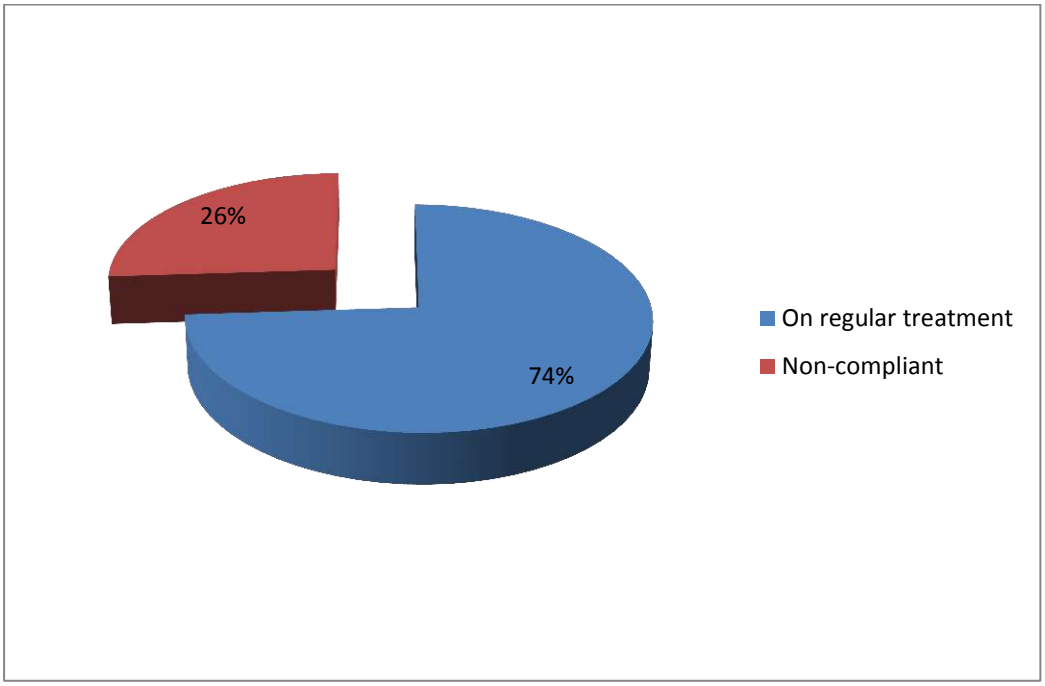

Figure 9: No. of Diabetes mellitus patients on regular treatment (n=43)

Table 10: Diabetes duration (n=43)

		No. of cases	Percentage
Duration of Diabetes Mellitus (In years)	< 5	11	25.6%
	5-10	16	37.2%
	> 10	16	37.2%
Total		43	100.0%

Most patients had diabetes mellitus between 5-10 years duration (37.2%) and same number of patients had diabetes mellitus for greater than 10 years (37.2%).

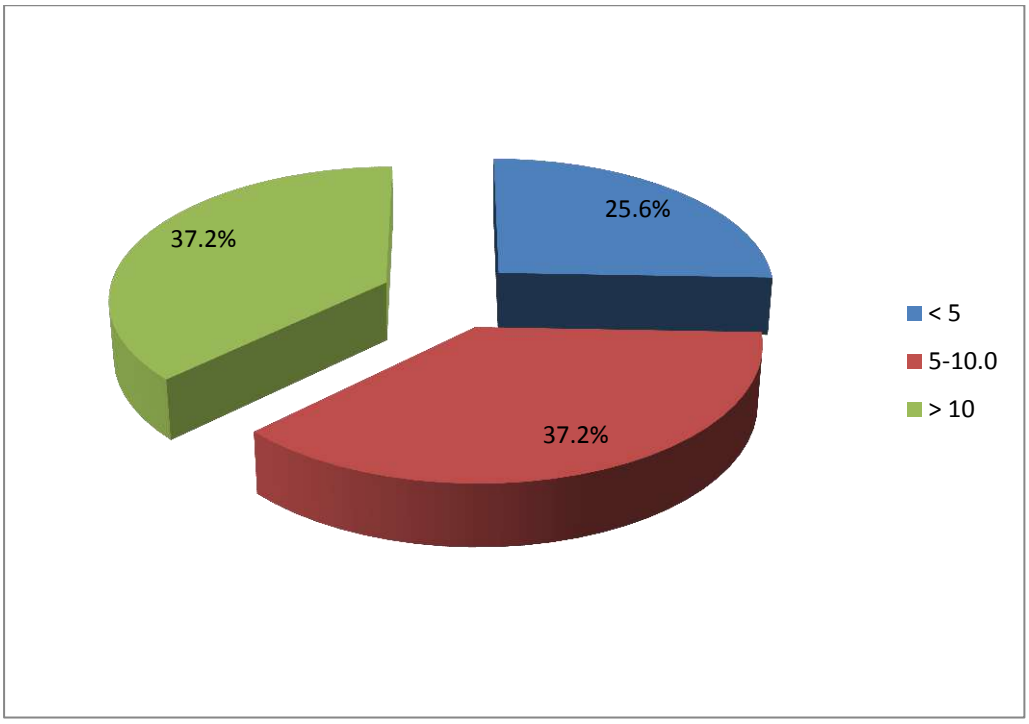

Figure 10: Diabetes duration (n=43)

Table 11: Diabetic retinopathy (n=43)

Fundus	No. of cases	Percentage
No diabetic retinopathy	26	60.5%
Mild non proliferative diabetic retinopathy	6	14.0%
Moderate non proliferative diabetic retinopathy	5	11.5%
Severe non proliferative diabetic retinopathy	6	14.0%
Total	43	100.0%

17 patients (39.5%) had non proliferative diabetic retinopathy. 6 patients (14%) had mild NPDR, 5 patients (11.5%)
had moderate NPDR and 6 patients (14.0%) had severe NPDR.
26 patients (60.5%) had no diabetic retinopathy.

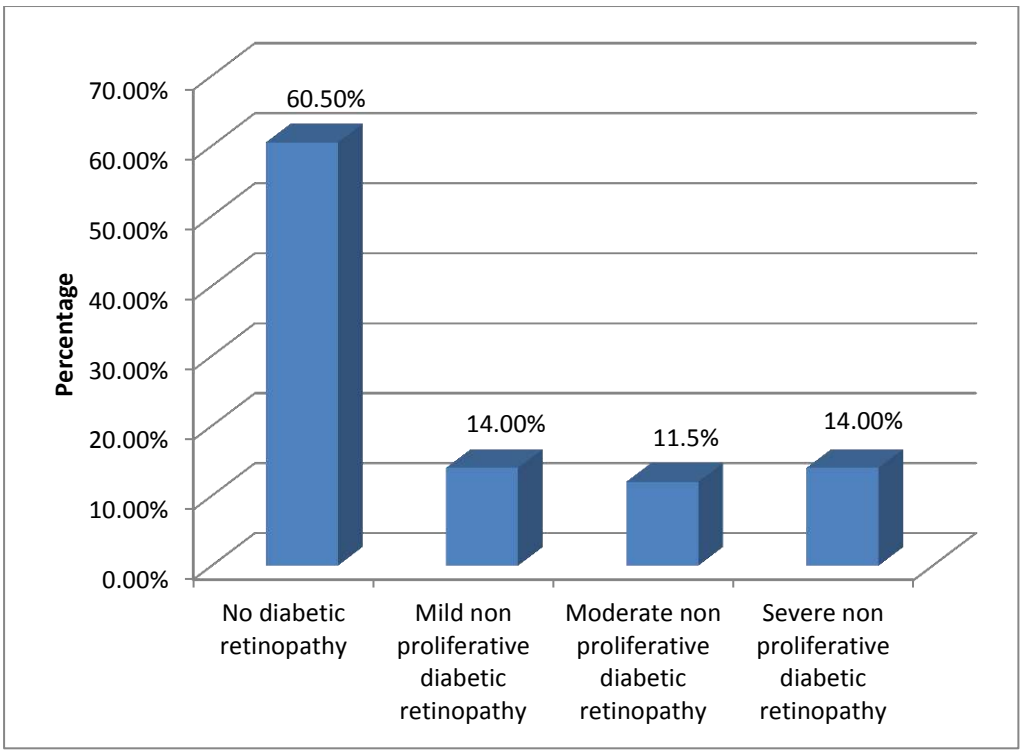

Figure 11: Diabetic retinopathy (n=43)

Table 12: Proteinuria (n=43)

Urine routine protein	No. of cases	Percentage
0	1	2.3%
1+	1	2.3%
2+	10	23.3%
3+	23	53.5%
4+	8	18.6%
Total	43	100.0%

42 out of the 43 patients had proteinuria while majority of patients (53.5%) had 3 + proteinuria in urine.

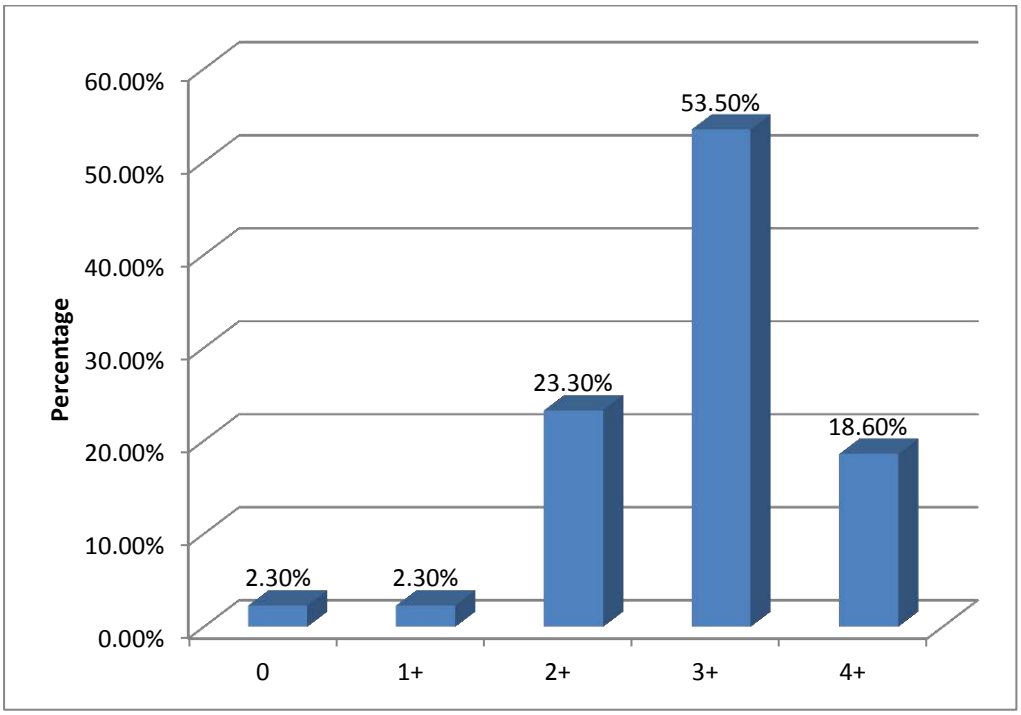

Figure 12: Proteinuria (n=43)

Table 13: Urine routine RBC (n=43)

Urine routine (rbc >5/hpf)	No. of cases	Percentage
0 to 3	32	74.4%
5 to 10	8	18.6%
11 to 25	1	2.3%
>50	2	4.7%
Total	43	100.0%

A total of 11 patients had significant microscopic hematuria.

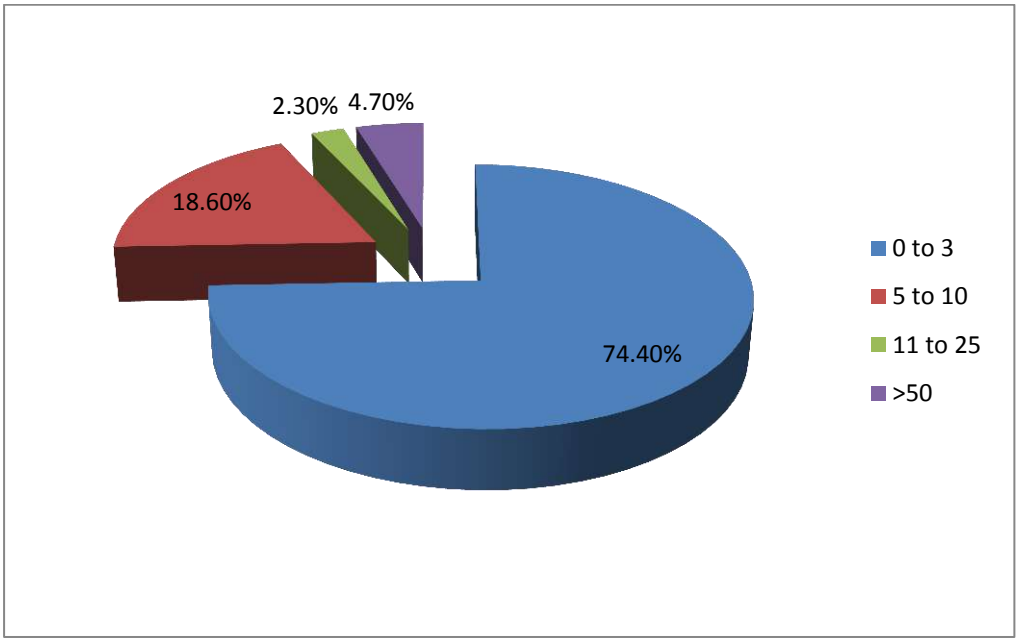

Figure 13: Urine routine RBC (n=43)

Table 14: UPCR (n=33)

UPCR	Total patients	Percentage
0.2 – 3.5	10	30.3%
>3.5	23	69.6%

A total of 33 patients had UPCR testing done. 10 patients had a UPCR between 0.2 – 3.5 and 23 patients had a UPCR >3.5 indicating nephrotic range proteinuria.

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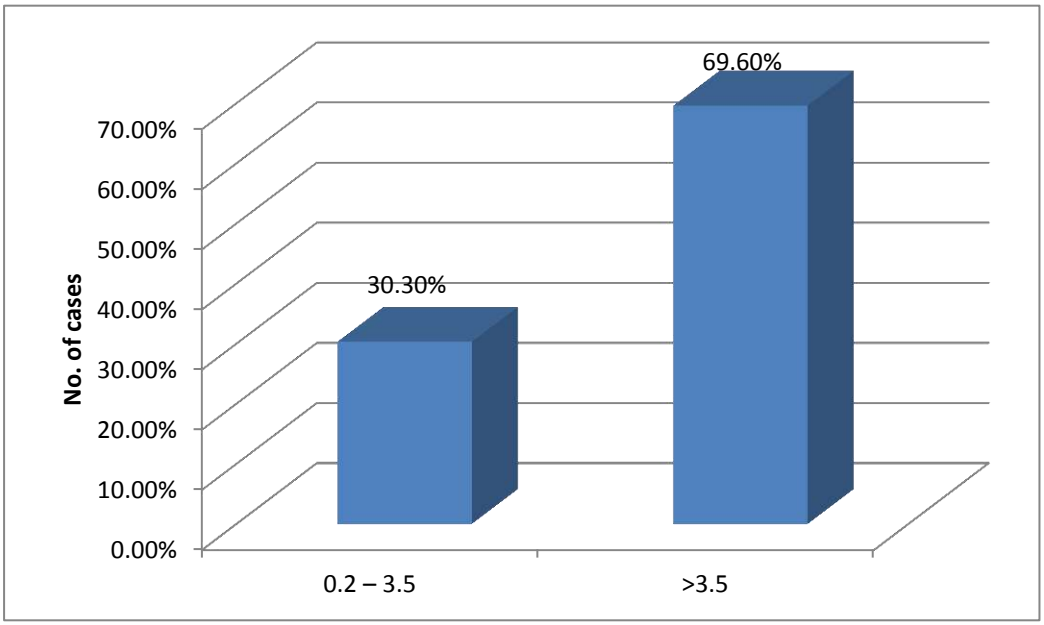


Figure 14: UPCR (n=33)

Table 15: USG abdomen (n=43)

Usg abdomen	No. of cases	Percentage
Not done	11	26.0%
Acute medical renal disease	12	27.9%
Bilateral renal parenchymal changes	14	32.5%
No significant finding	6	14.0%
Total	43	100.0%

14 out of 43 patients (34.9%) had bilateral renal parenchymal changes, 12 patients showed (27.9%) acute medical renal disease and 6 patients had no significant findings on sonography.

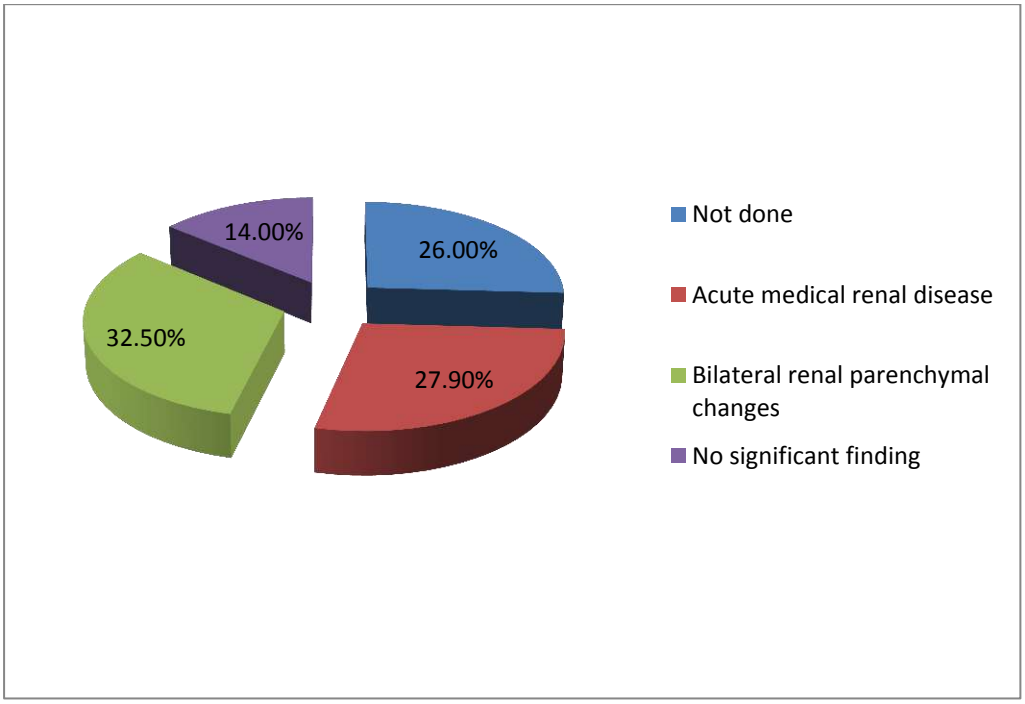


Figure 15: USG abdomen (n=43)

Patients having features of both non-diabetic kidney disease and diabetic kidney disease (mixed renal disease) were added to the non-diabetic kidney disease group for statistical purposes. Thus, there were 28 patients of non-diabetic kidney disease, and 15 patients of diabetic kidney disease.

Table 16: Histopathological impression (n=43)

Histopathology impression	No. of cases	Percentage
Diabetic kidney disease	15	35.0%
Non diabetic kidney disease	24	56.0%
Mixed	4	9.3%
Total	43	100.0%

Renal biopsy revealed 24 out of 43 patients having pure non-diabetic kidney disease (56.0%). 15 patients had pure diabetic kidney disease (35%) and 4 patients had a mixed picture of non-diabetic kidney disease (9.3%) plus diabetic kidney disease.

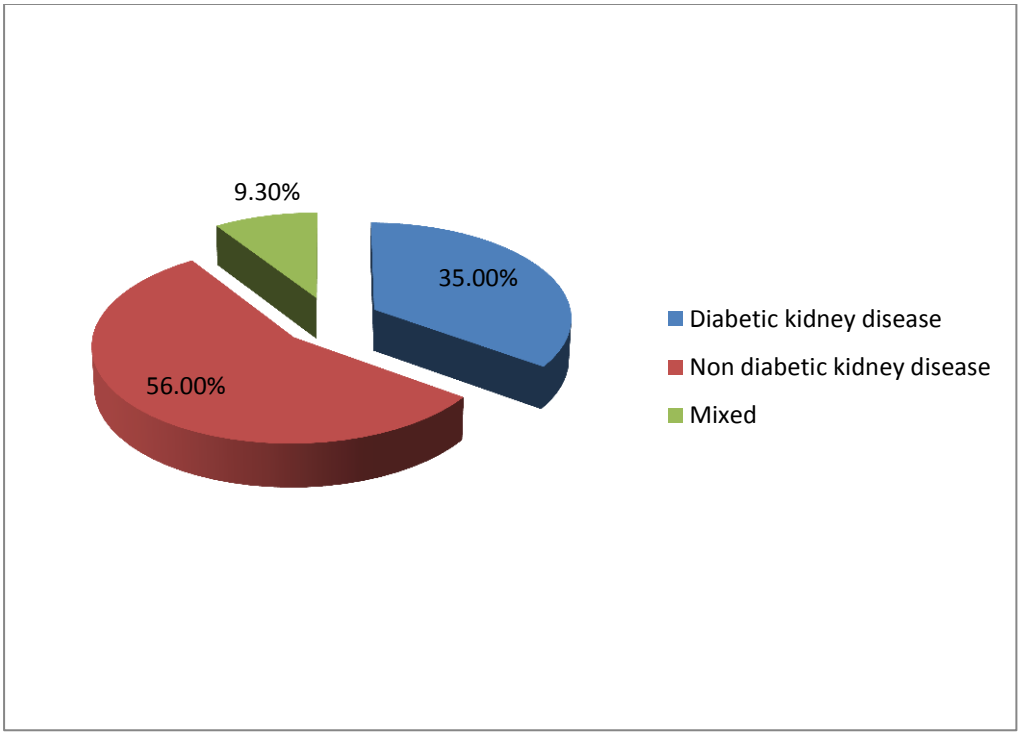


Figure 16: Histopathological impression (n=43)

Table 17: Histopathological report and immuno-florescence

Renal biopsy	Cases	Percentage%	Direct immuno-florescence
Diabetic nephropathy	15	35%	No glomerular deposits
Mixed (diabetic nephropathy + non-diabetic kidney disease) a) Diabetic nephropathy with features of acute pyelonephritis	1	2.3%	No glomerular deposits
b) Diabetic nephropathy with features of acute tubulo-interstitial nephritis	2	4.6%	No glomerular deposits
c) Focal proliferative glomerular nephritis. Diabetic nephropathy and acute tubulo-interstitial nephritis.	1	2.3%	No glomerular deposits
IgA Nephropathy	3	7.0%	IgA - mesangial positivity +3. Kappa - mesangial positivity +2. Lambda mesangial +1
Crescentic glomerulonephritis	3	7.0%	Dif core shows 5 sclerosed glomeruli
Focal Proliferative Glomerulonephritis	3	7.0%	No glomerular deposits
Membranous Nephropathy	2	4.7%	Granular positivity of igg +3 and c3c +2 noted along the glomerular capillary loops
Post-infectious glomerulonephritis	1	2.3%	Strong 2+ coarse granular staining for c3c and 1+ staining for iga
Focal segmental glomerulosclerosis	1	2.3%	6 viable and 4 sclerosed glomeruli
Renal amyloidosis	1	2.3%	Deposits are positive for kappa (2+)
Lupus nephritis	1	2.3%	Full house positivity
Acute tubular Necrosis	2	4.7%	No glomerular deposits
Acute interstitial Nephritis	5	12%	No glomerular deposits
Chronic interstitial nephritis	1	2.3%	IgG deposits tubular basement membrane
Acute pyelonephritis	1	2.3%	No glomerular deposits
Total	43	100%	

The most common pathology in NDKD was AIN in 5 patients (16.7%) followed by IgA nephropathy, Crescentic glomerulonephritis and Focal proliferative glomerulonephritis accounting for 3 patients each. Diabetic nephropathy was seen in 15 patients (35.0%) and 4 patients had mixed renal disease (9.3%).

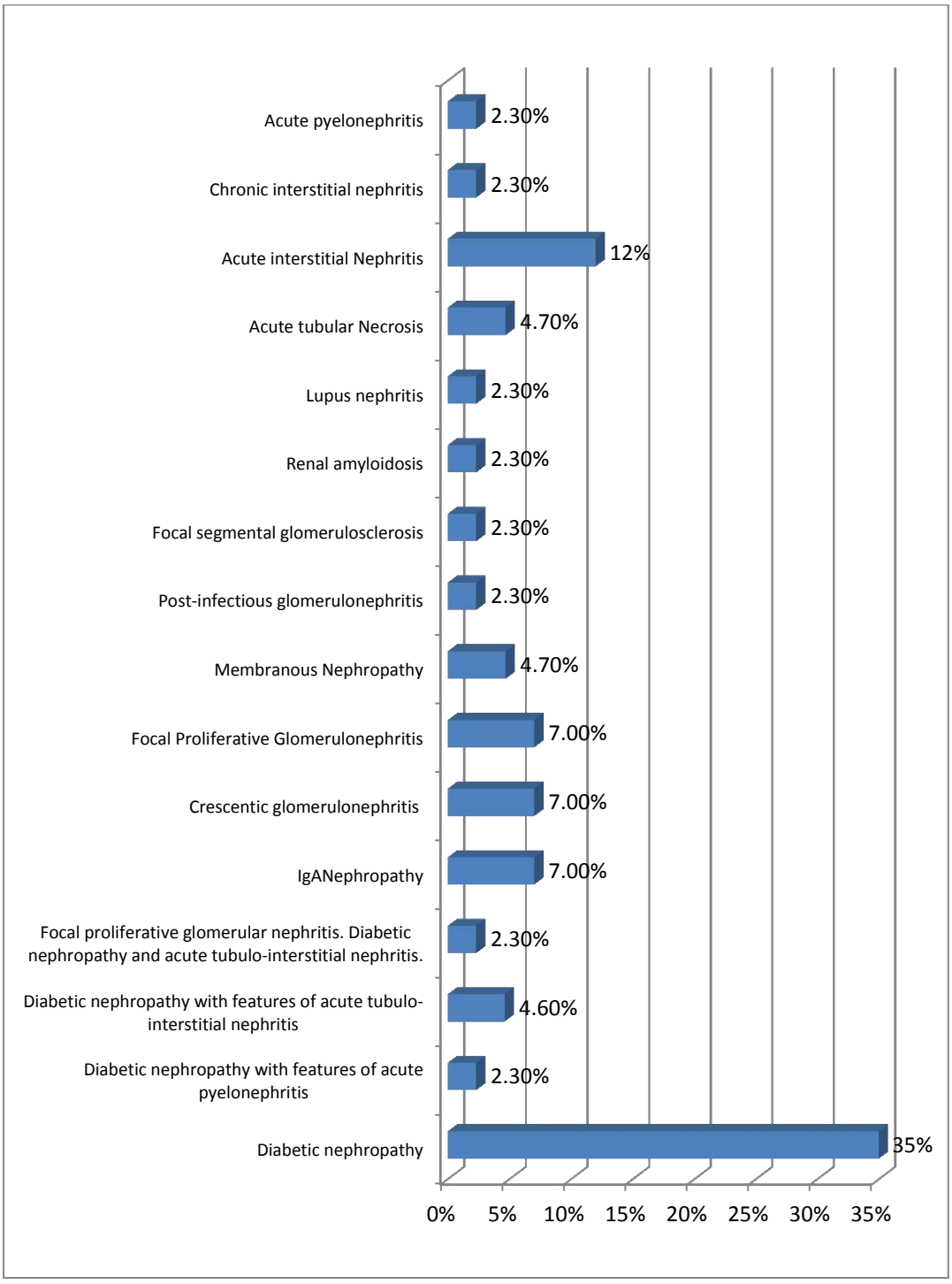
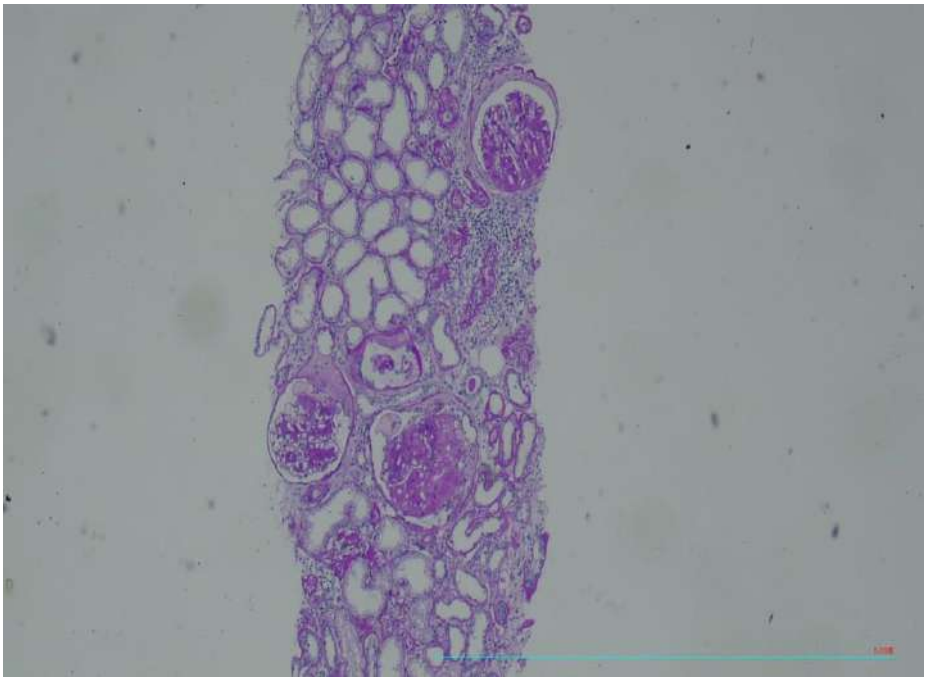
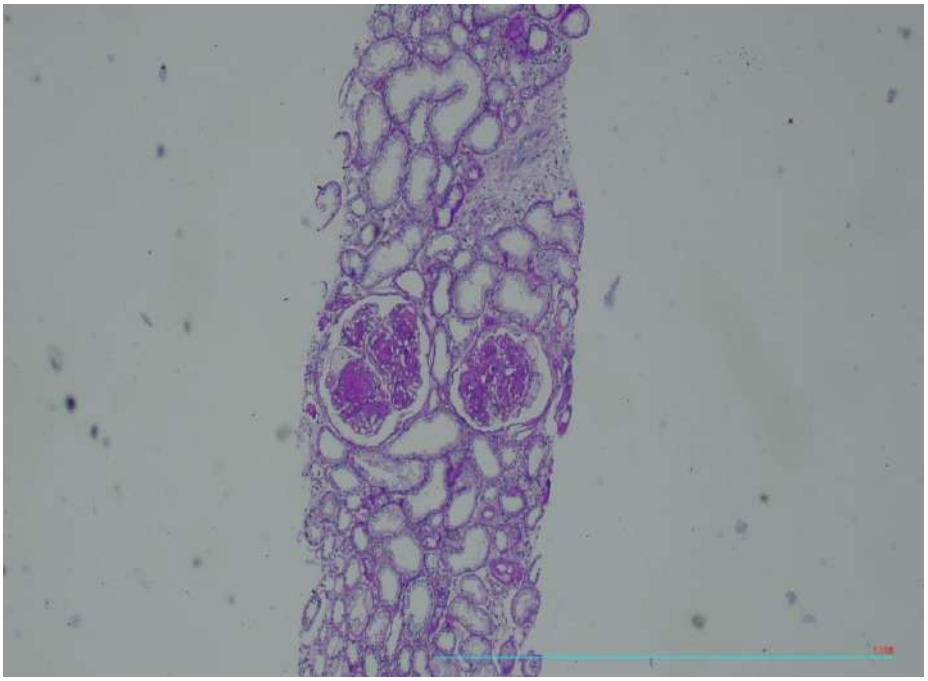
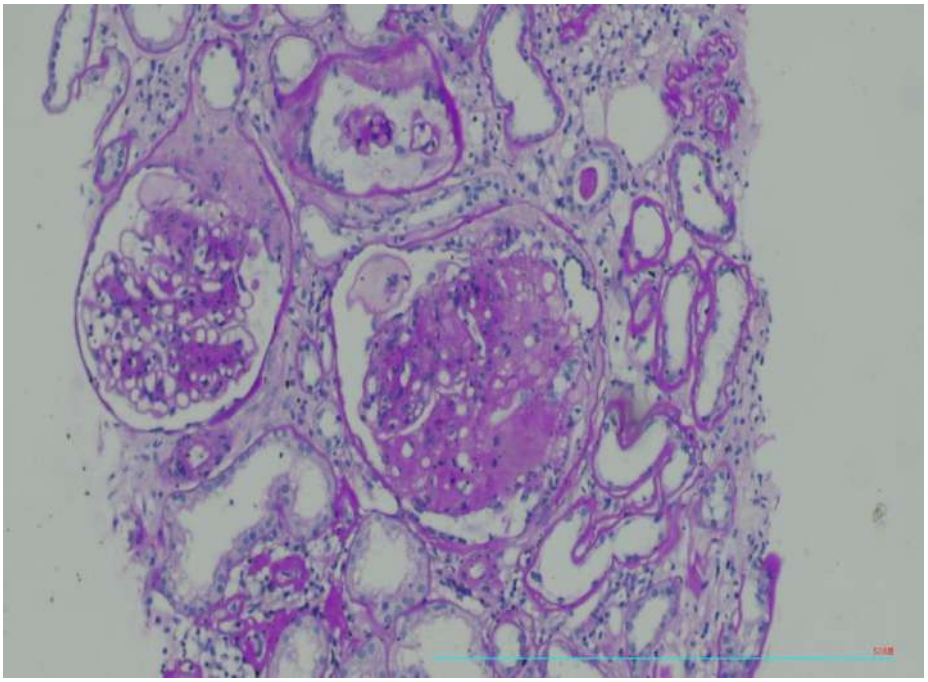


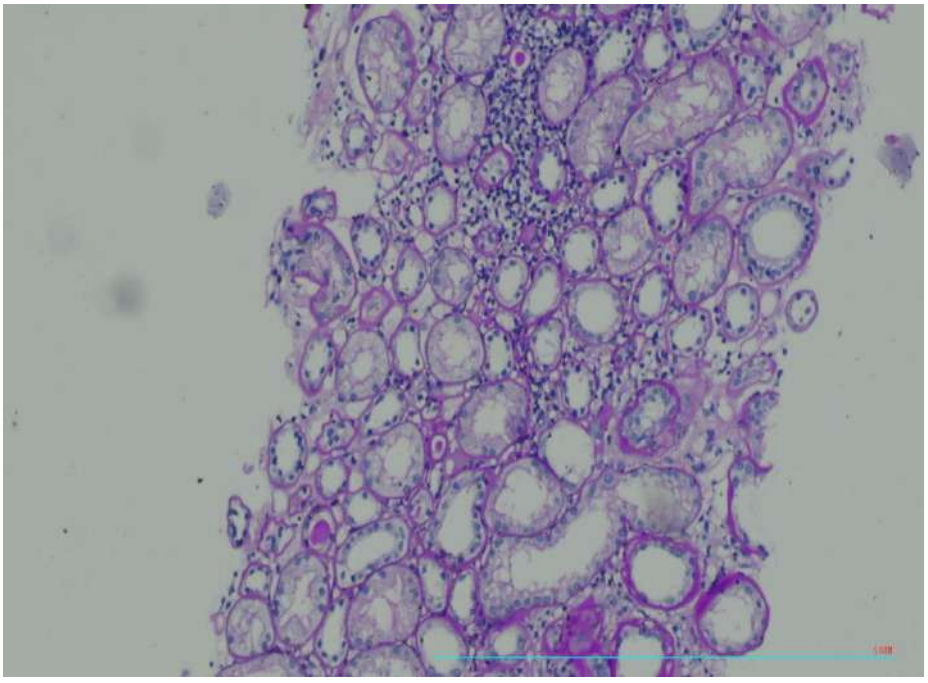
Figure 17: Histopathological report and immuno-florescence

Pic 1: Acute Interstitial Nephritis with diabtic Nephropathy

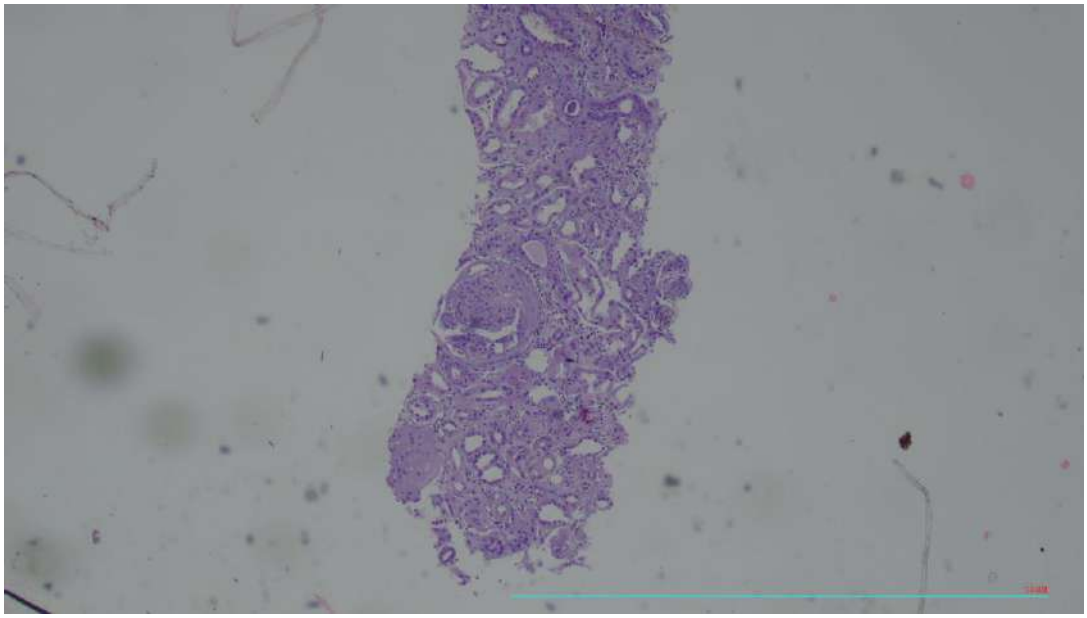


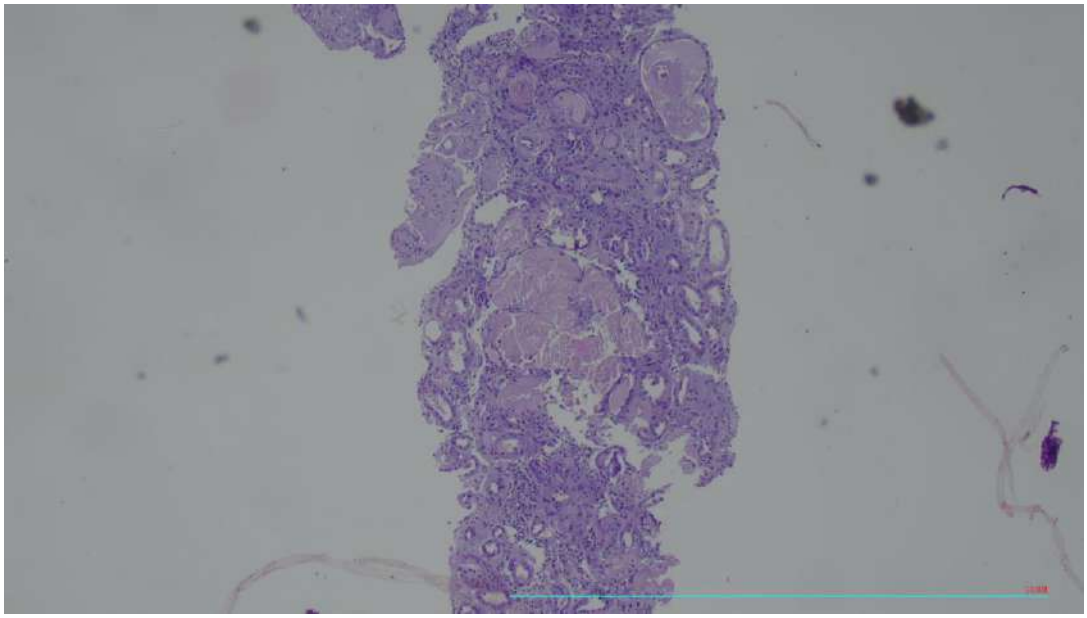


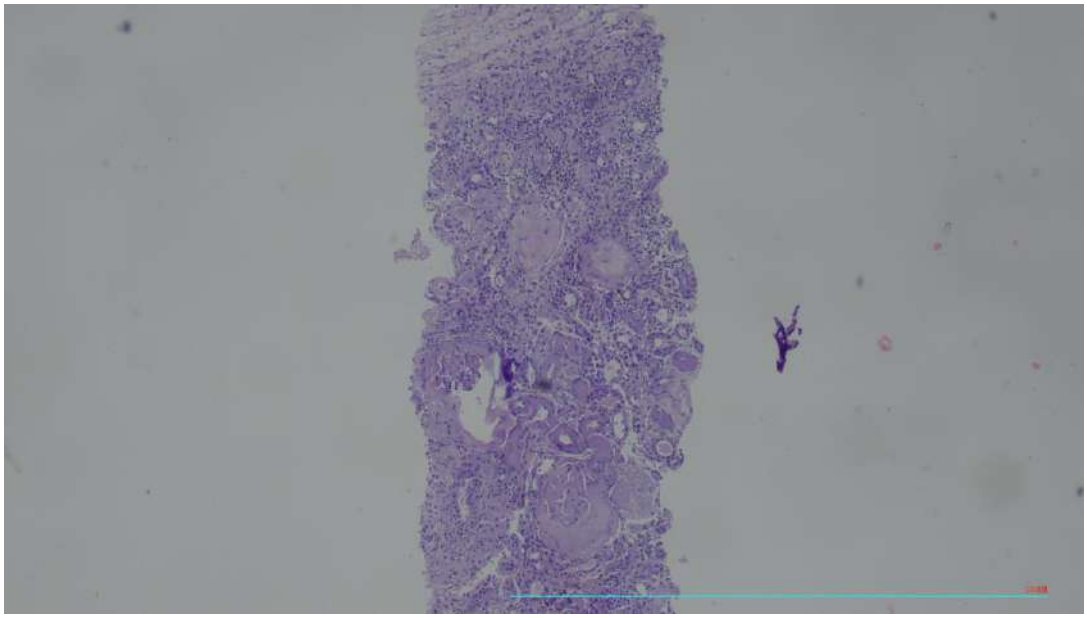


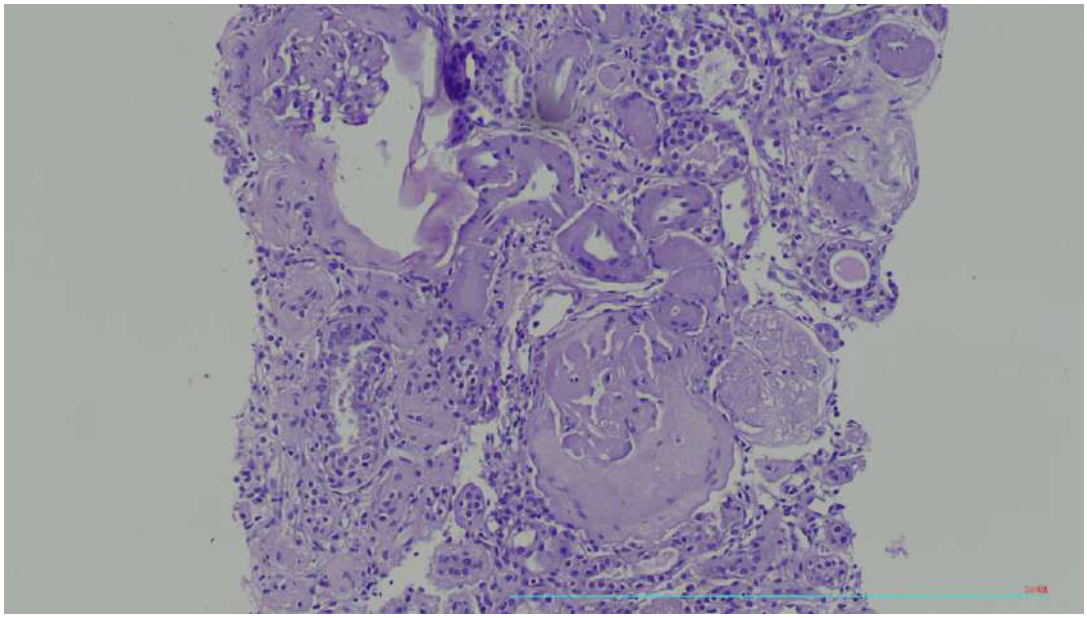


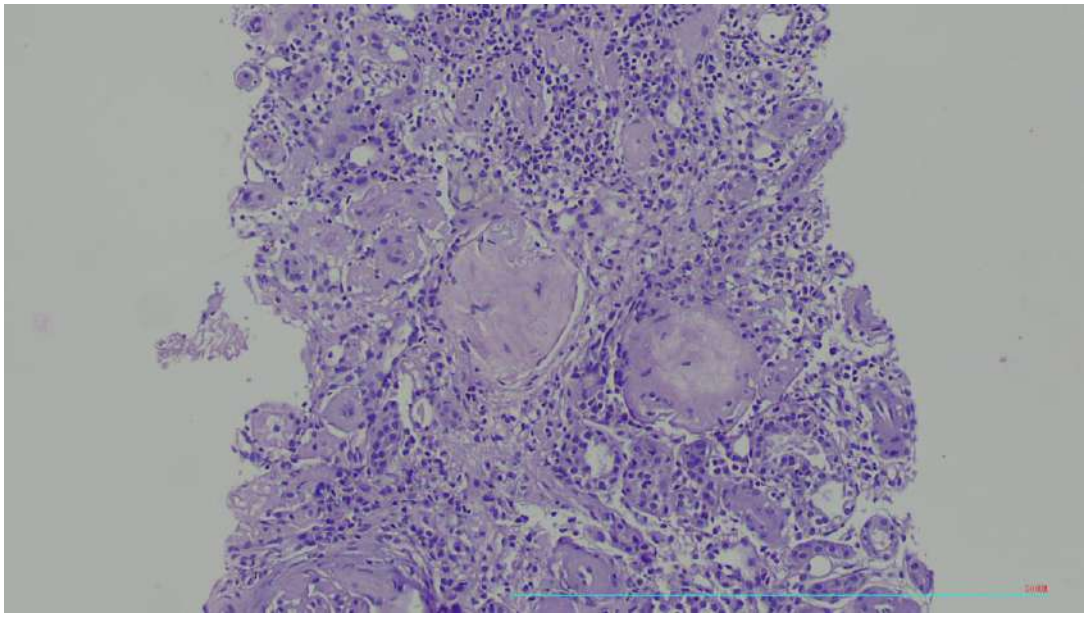
Pic 2: Post infectious glomerulonephritis with Acute interstitial nephritis

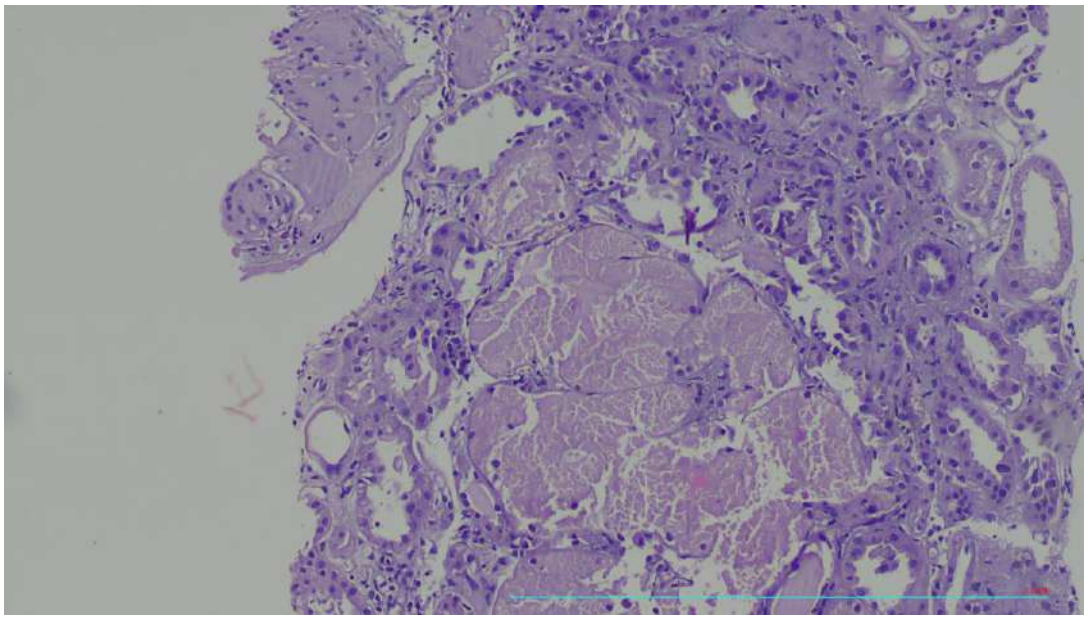




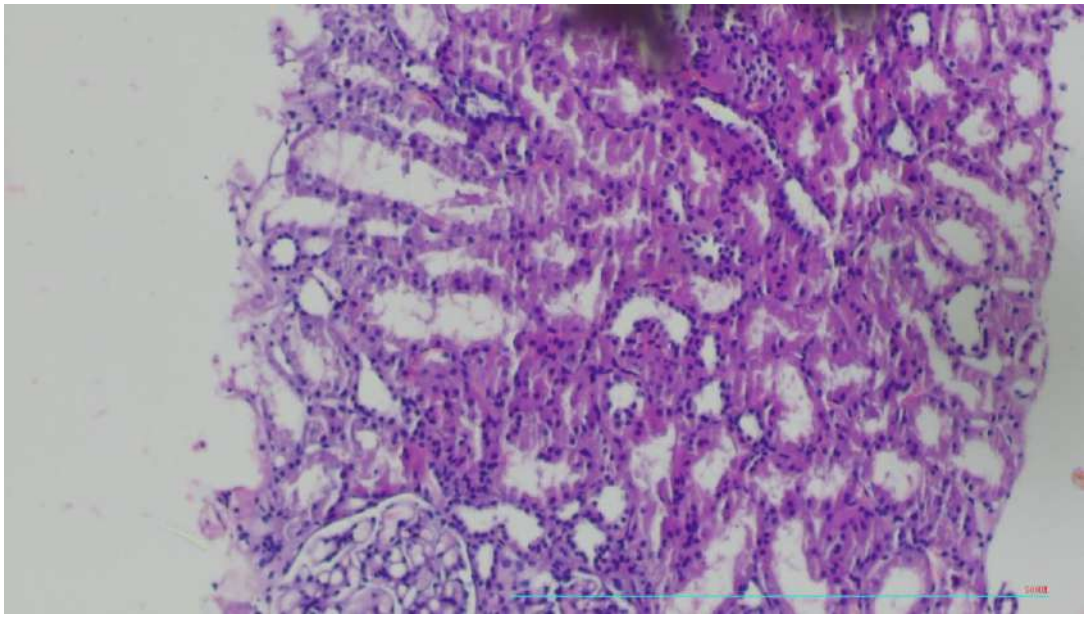


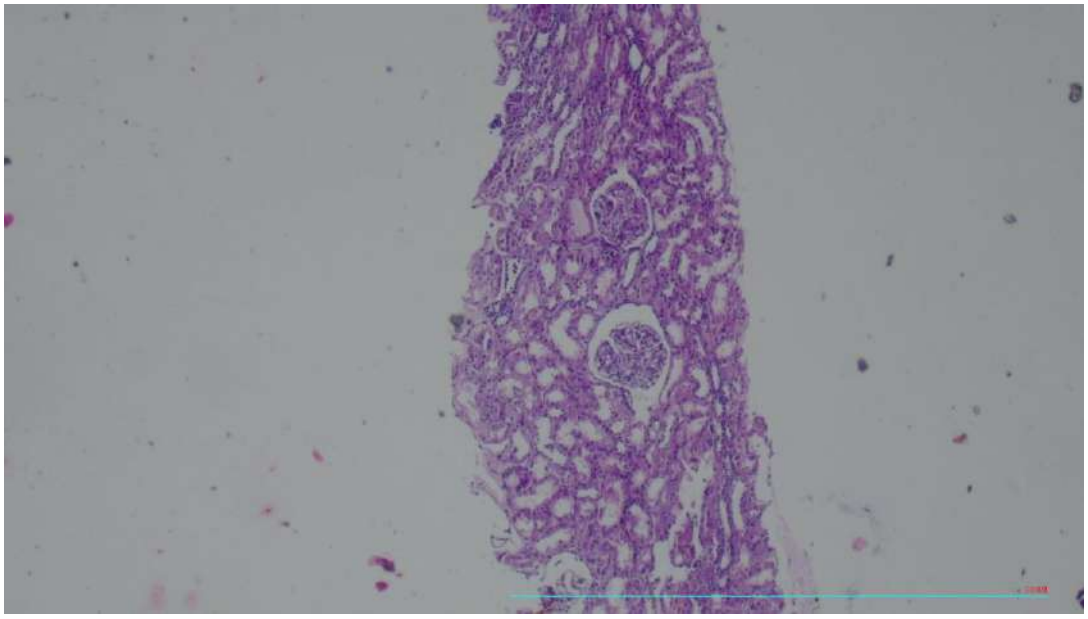


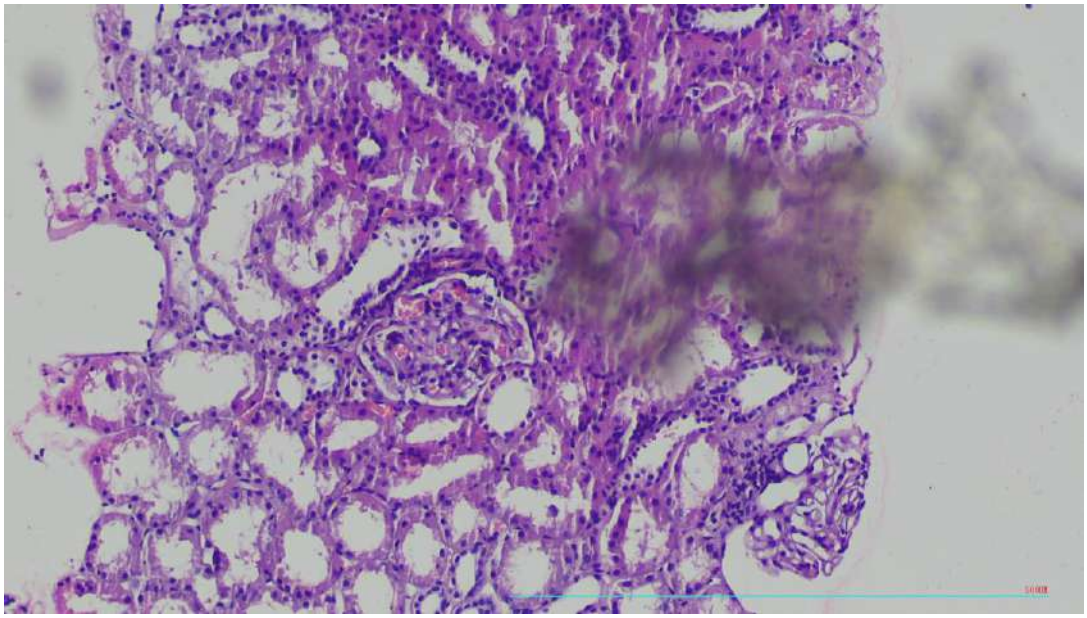


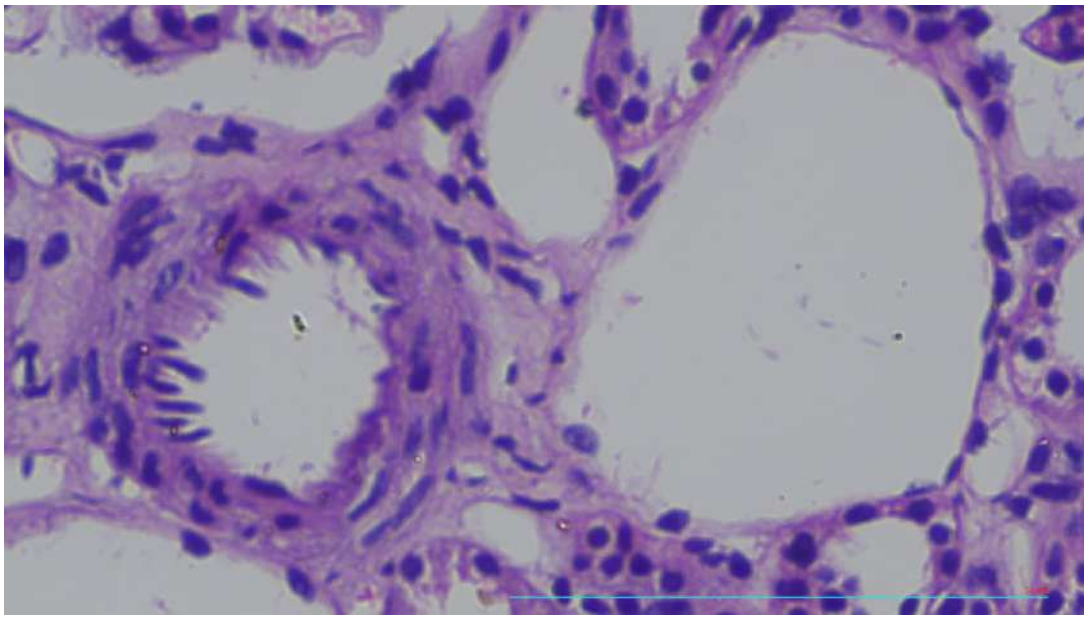


Pic 3: Membranous Nephropathy

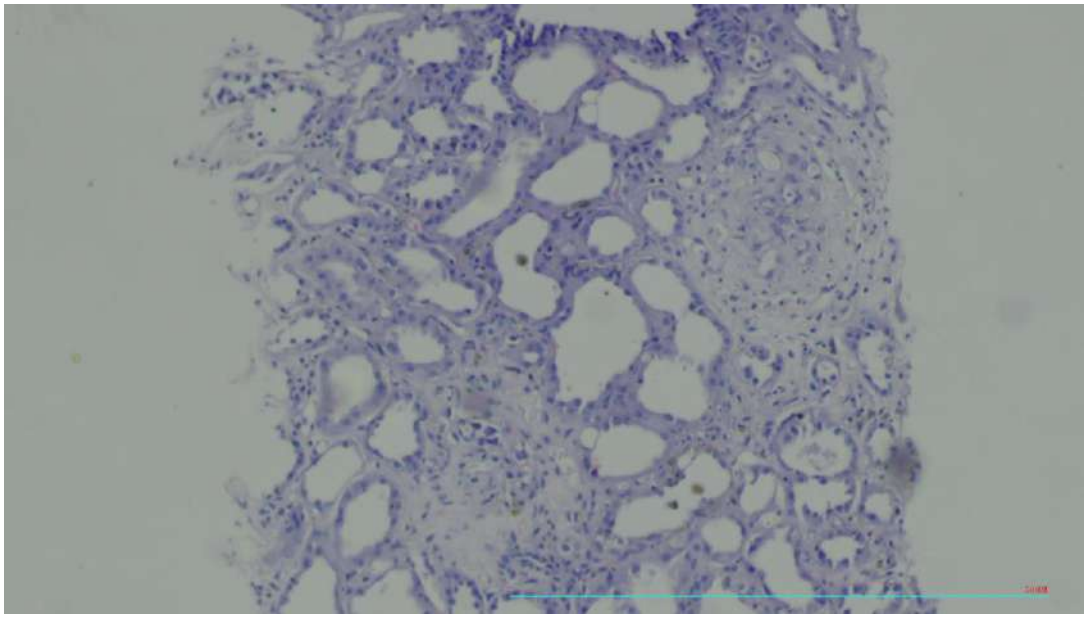


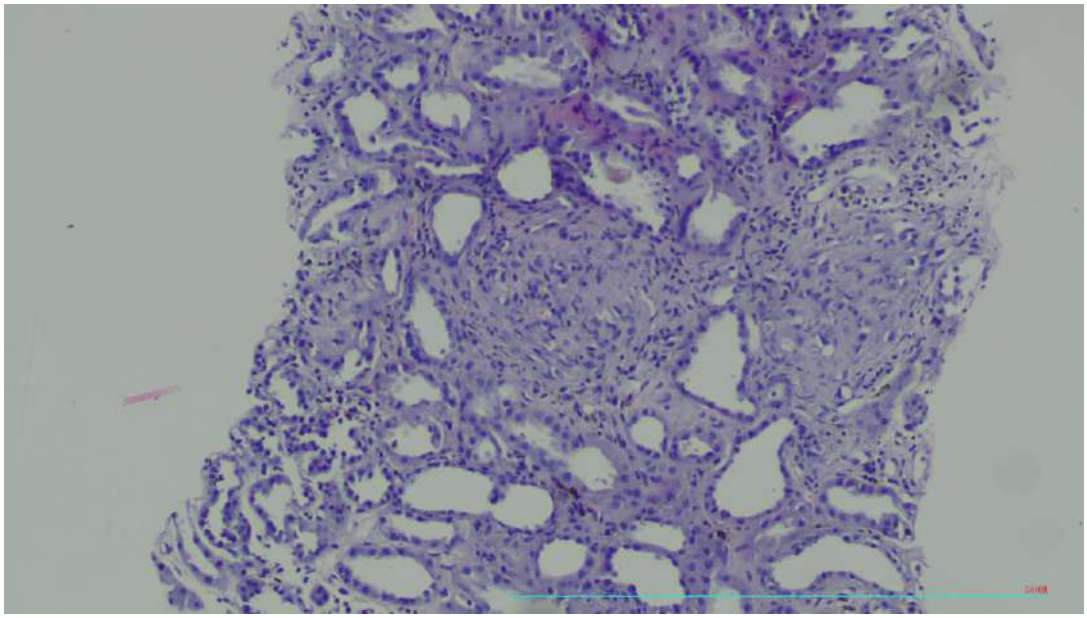


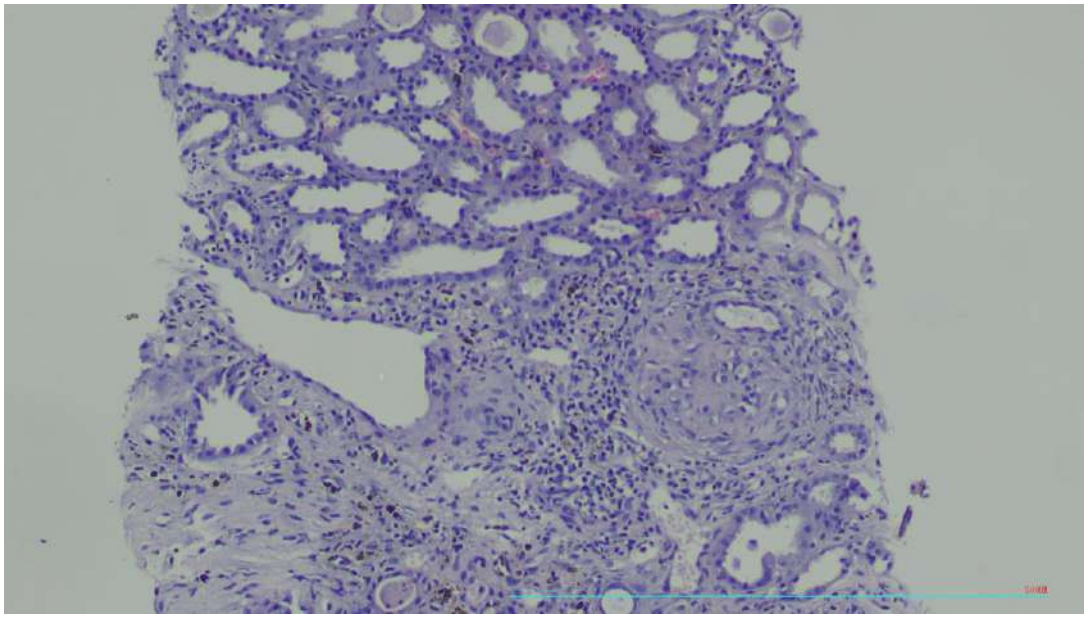


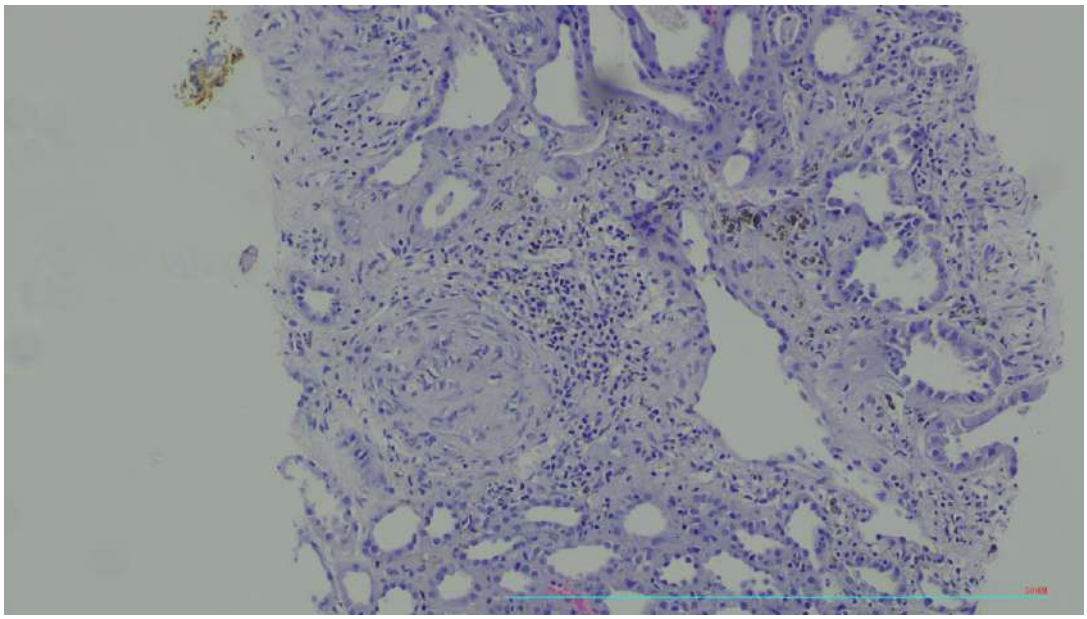


Pic 4: Crescentic glomerulonephritis

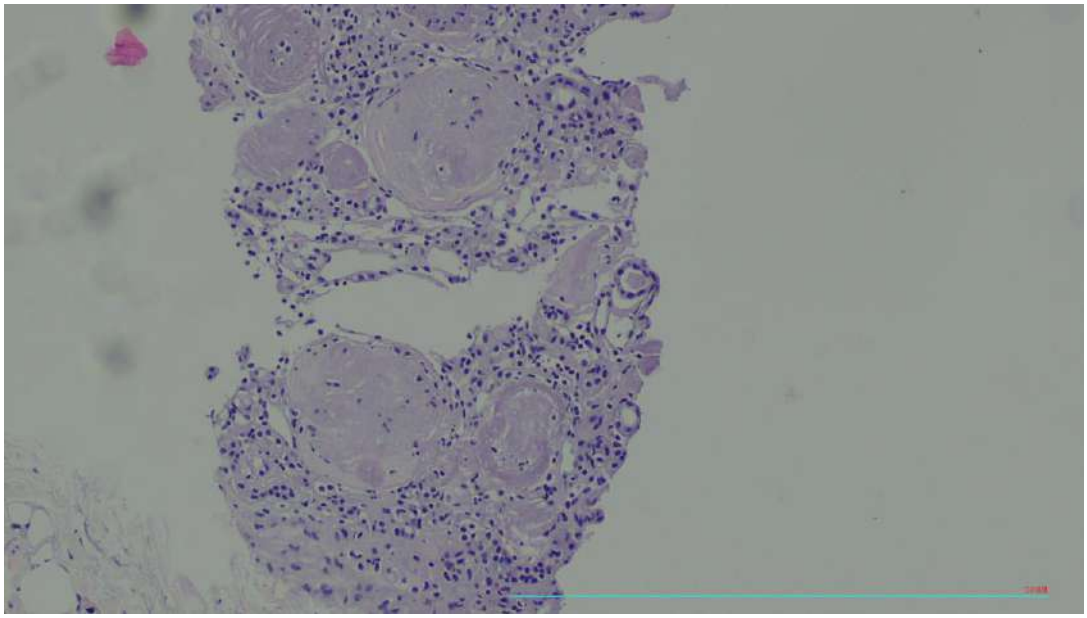


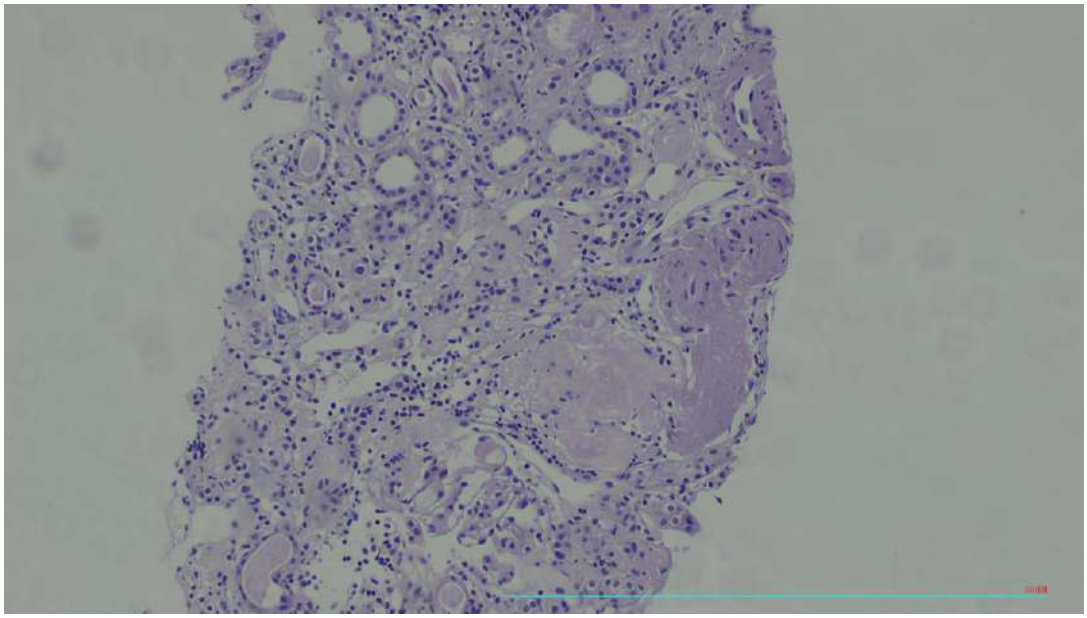


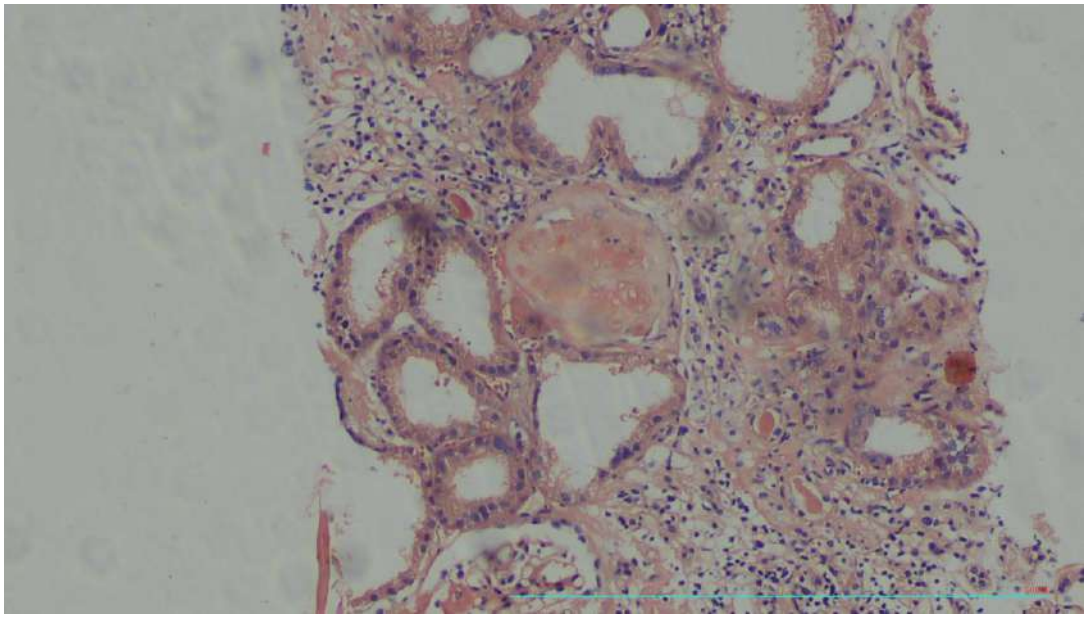


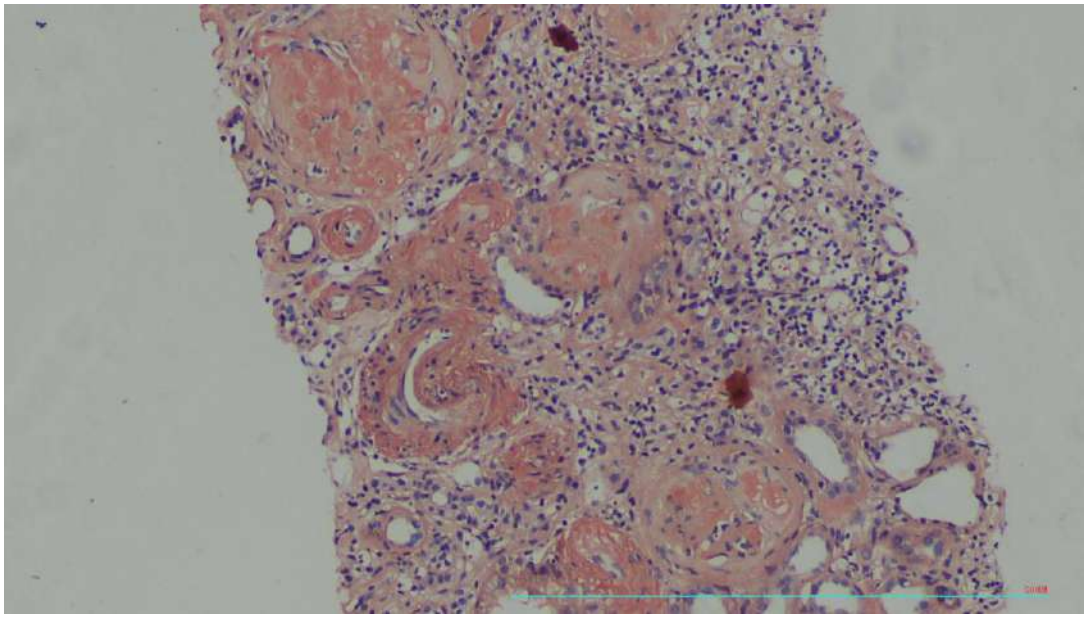


Pic 5: Renal amyloidosis



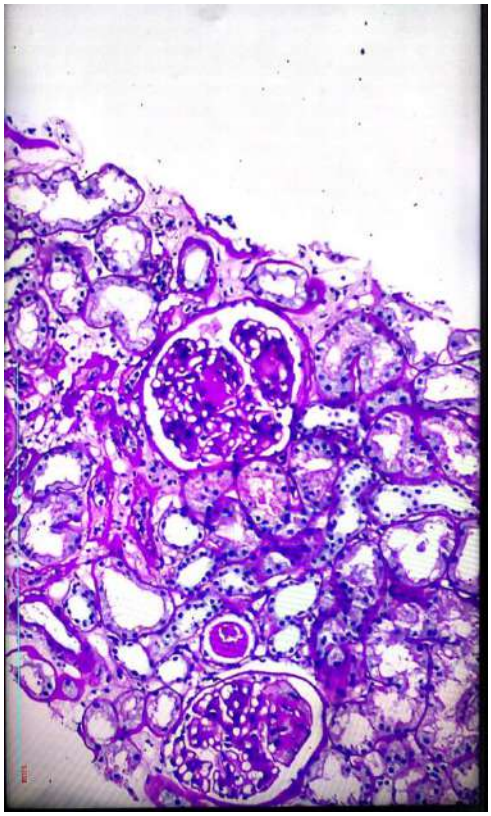


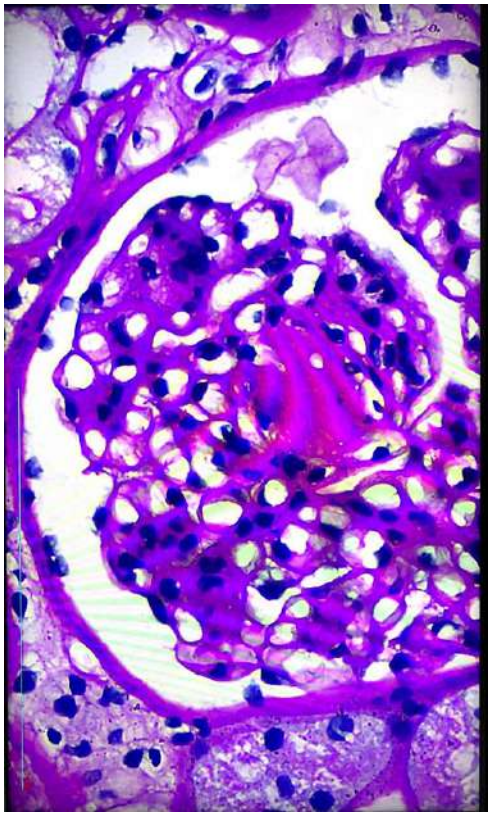




PIC 6: DIABETIC KIDNEY DISEASE

PAS STAIN





SILVER STAIN

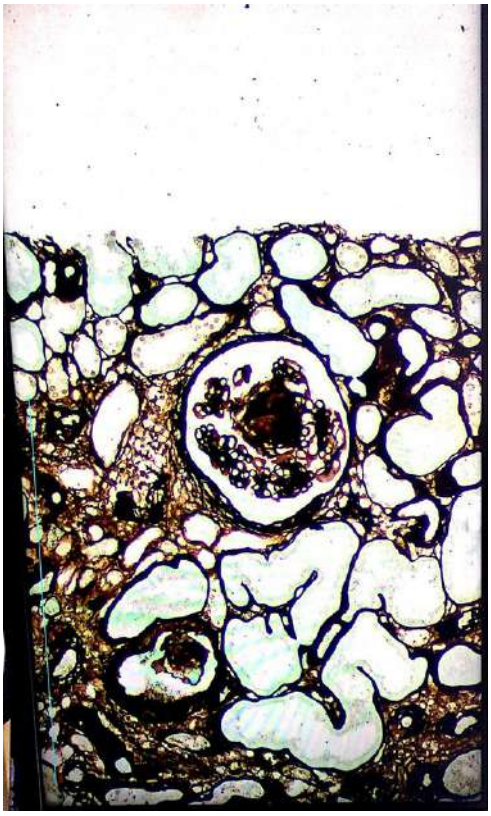


Table 18: Age groups in non-diabetic kidney disease and diabetic kidney disease (n=43)

		DKD		NDKD		Total	Chi-square value	P-value
Age group	< 40	1	6.7%	3	10.7%	4	1.206	0.877
	41-50	2	13.3%	6	21.4%	8		
	51-60	5	33.3%	8	28.6%	13		
	61-70	4	26.7%	8	28.6%	12		
	> 70	3	20.0%	3	10.7%	6		
Total		15	100.0%	28	100.0%	43		

Non-diabetic kidney disease was most frequently diagnosed in the 51–60 and 61-70 age group, whereas age group 51-60 was most common for diabetic kidney disease followed by 61–70-year age group as the second commonest.

The above table highlights that NDKD was more common in the 41-50 years age group as compared to the DKD group. (21.4% vs 13.3%)

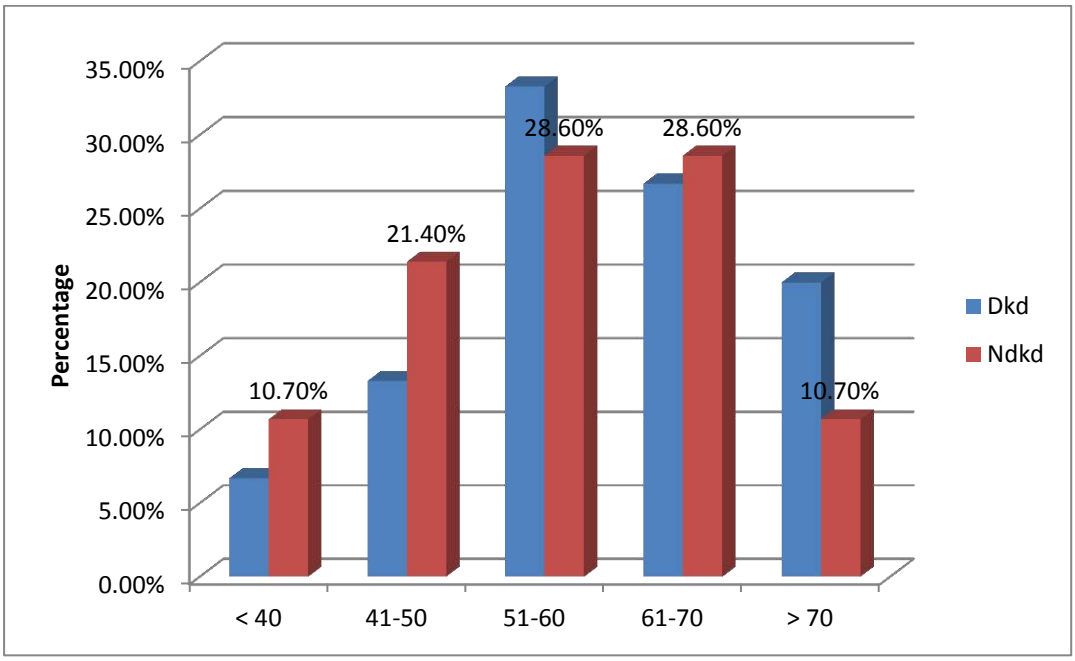


Figure 18: Age groups in non-diabetic kidney disease and diabetic kidney disease (n=43)

Table 19: Mean age in non-diabetic kidney disease and diabetic kidney disease (n=43)

Ndkd	DKD		NDKD		Z	P-value
	Mean	Sd	Mean	Sd		
Age	59.60	11.42	55.86	11.29	1.032	0.308

The mean age in non-diabetic kidney disease was 55.86 ± 11.29 which was less than the mean age in diabetic kidney disease was 59.60 ± 11.42 though it was not statistically significant.

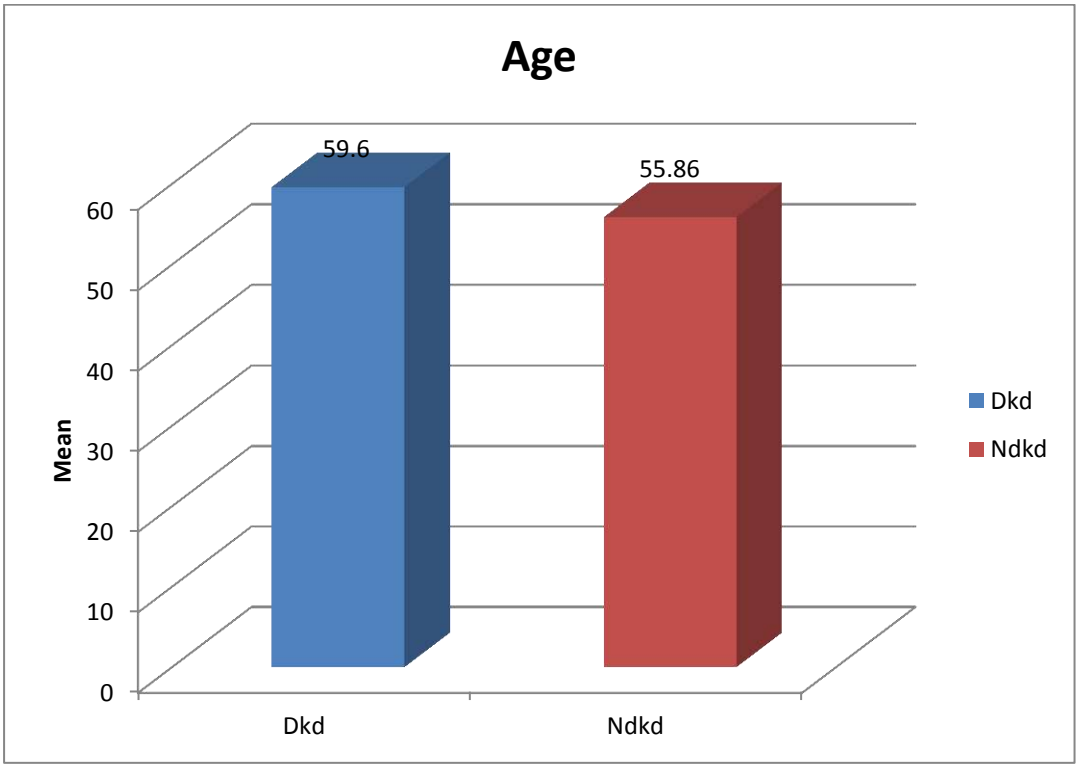


Figure 19: Mean age in non-diabetic kidney disease and diabetic kidney disease (n=43)

Table 20: Gender (n=43)

		DKD		NDKD		Total	Chi-square value	P-value
Sex	Female	6	40.0%	10	35.7%	16	0.077	0.782
	Male	9	60.0%	18	64.3%	27		
Total		15	100.0%	28	100.0%	43		

Diabetic kidney disease and non-diabetic kidney disease was more common in males representing 60.0% and 64.3% respectively. Male to female ratio for DKD was 1:1.5 vs 1:1.8 in the NDKD group.

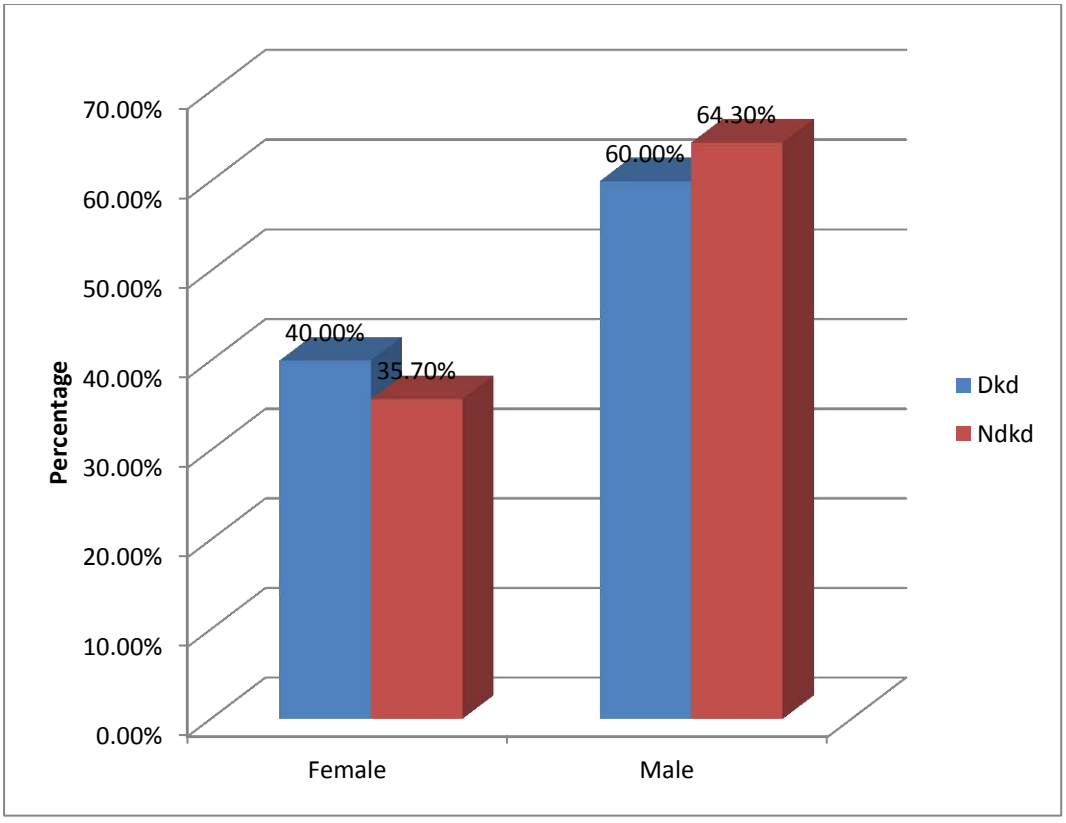


Figure 20: Gender (n=43)

Table 21: History of presenting illness (n=43)

History of presenting illness		DKD		NDKD		Total
Dyspnoea	Present	7	46.7%	9	32.1%	16
Decreased urine output	Present	5	33.3%	12	42.9%	17
Facial puffiness	Present	3	20.0%	9	32.1%	12
Oedema feet	Present	12	80.0%	16	57.1%	28
Haematuria	Present	0	0.0%	2	7.1%	2
Total		15	100.0%	28	100.0%	43

Majority of patients in both groups had oedema of the feet as a major presenting symptom. Facial puffiness was more common in NDKD in comparison to the DKD group. 9 patients in the NDKD group had dyspnea as a presenting symptom vs 7 patients in the DKD group. 5 patients had decreased urine output in the DKD group vs 12 patients in the NDKD group.

Table 22: Drug abuse in non-diabetic kidney disease and diabetic kidney disease (n=43)

		DKD		NDKD		Total	Chi-square value	P-value
Smoking (yes/no)	No	11	73.3%	22	78.6%	33	0.150	0.719
	Yes	4	26.7%	6	21.4%	10		
Total		15	100.0%	28	100.0%	43		
Alcohol (yes/no)	No	11	73.3%	23	82.1%	34	0.458	0.696
	Yes	4	26.7%	5	17.9%	9		
Total		15	100.0%	28	100.0%	43		
Any other drug abuse (yes/no)	No	15	100.0%	28	100.0%	43		
Total		15	100.0%	28	100.0%	43		

In patients with diabetic kidney disease, 4 out of 15 patients were smokers (26.7%) and 4 patients (26.7%) consumed alcohol regularly.

In the non-diabetic kidney disease group, 6 patients out of 28 (21.4%) were smokers and 5 patients (17.9%) consumed alcohol regularly. The findings are similar in both the groups.

Table 23: Drug intake and histopathological diagnosis in NDKD (n=8)

NSAIDS/ Indigenous meds	Patient number	Percentage %	Histopathology	Cases
DKD	2	13.3%	Diabetic Nephropathy	2
NDKD	6	21.4%	• Diffuse proliferative glomerulo-nephritis • Focal proliferative glomerulo-nephritis • Acute tubule-interstitial nephritis • Diabetic nephropathy with acute interstitial nephritis	1 1 2 2

A total of 8 patients were taking NSAID/Indigenous medication among our study group out of which 6 were in the NDKD group while 2 were in the DKD group. We analyzed the histopathological diagnosis in these patients and found AIN and DN with AIN were the most common pathology in the NDKD group.

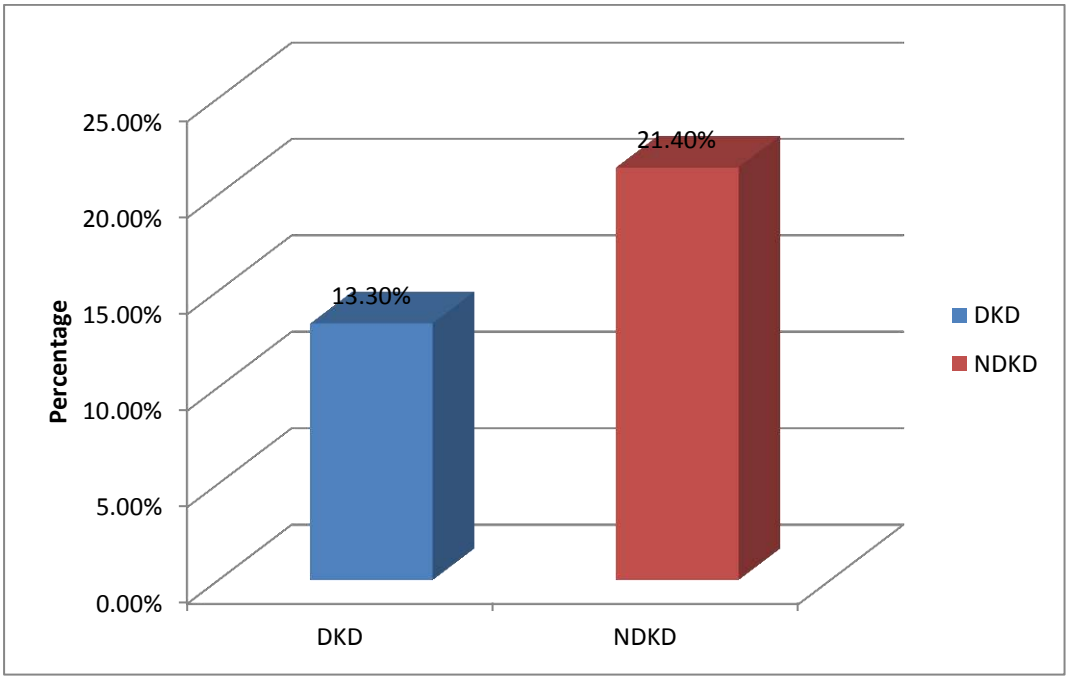


Figure 21: Drug intake and histopathological diagnosis in NDKD (n=8)

Table 24: Hypertension in non-diabetic kidney disease and diabetic kidney disease (n=43)

		DKD		NDKD		Total	Chi-square value	P-value
HTN	No	1	6.7%	8	28.6%	9	2.832	0.129
	Yes	14	93.3%	20	71.4%	34		
Total		15	100.0%	28	100.0%	43		

Number of patients with hypertension were more in the DKD group (93.3%) vs NDKD group (71.4%). It was however not statistically significant.

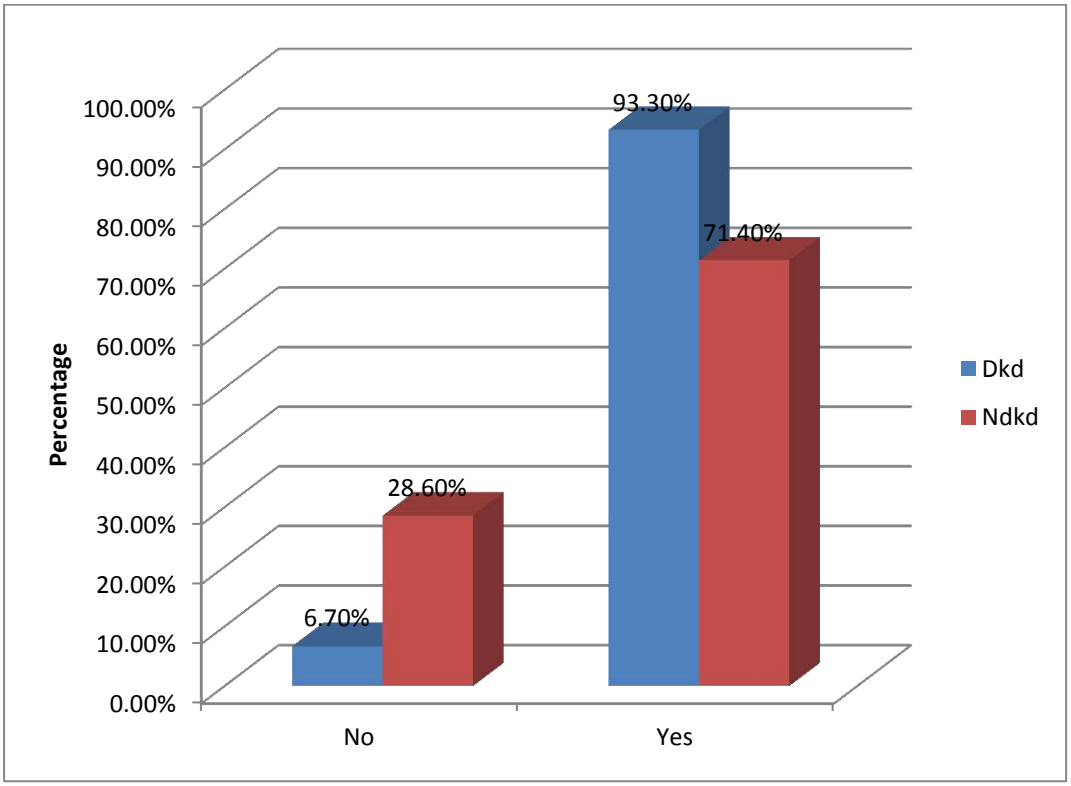


Figure 22: Hypertension in non-diabetic kidney disease and diabetic kidney disease (n=43)

Table 25: Duration of Hypertension, Diabetes and BMI Non-diabetic kidney disease and diabetic kidney disease (n=43)

Ndkd/dkd	DKD		NDKD		Z	P-value
	Mean	SD	Mean	SD		
Diabetes mellitus number of years:	12.03	6.84	9.60	6.03	1.201	0.236
Hypertension number of years:	8.07	4.65	4.98	2.86	2.407	0.022
Bmi (kg/m2)	24.33	2.35	24.36	2.47	0.031	0.976

Hypertension duration in non-diabetic kidney disease group was 4.98 ± 2.86 years and in the diabetic kidney disease group it was 8.07± 4.65 years which was statistically significant.

Duration of diabetes in the non-diabetic kidney disease group was less than the diabetic kidney disease group but was statistically not significant.

BMI in the diabetic kidney disease group and non-diabetic kidney disease group were almost identical.

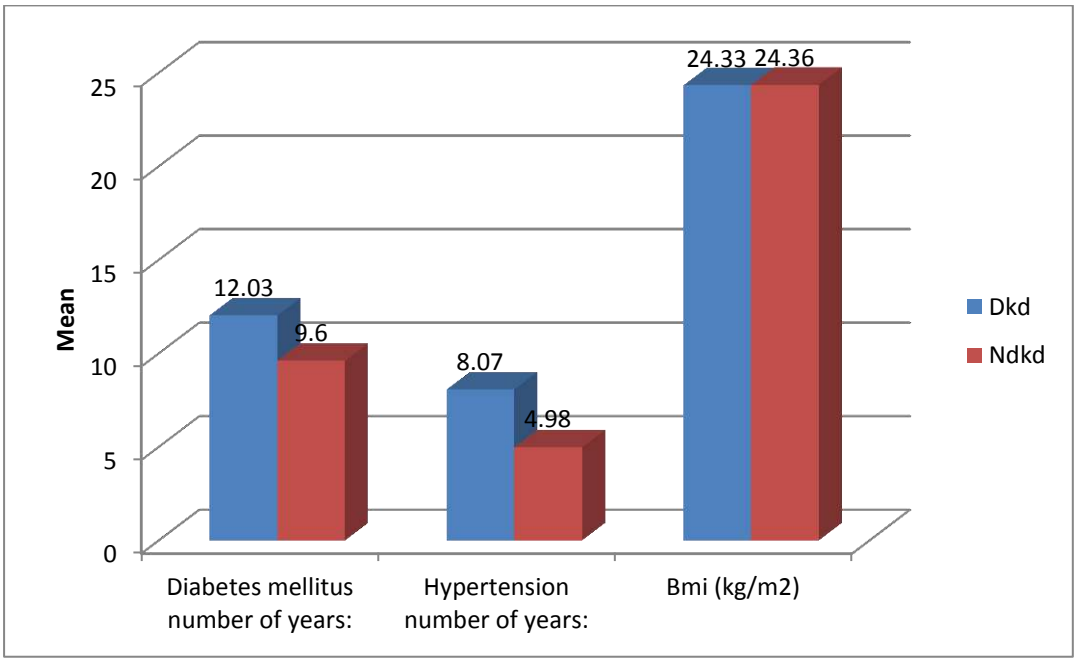


Figure 23: Duration of Hypertension, Diabetes and BMI Non-diabetic kidney disease and diabetic kidney disease (n=43)

Table 26: Diabetes duration in NDKD and DKD (n=43)

	Number of Years	DKD	%	NDKD	%	Total	Chi-square value	p-value
Duration of Diabetes Mellitus	< 5	3	20.0%	8	28.6%	11	0.927	0.926
	5-10	5	33.3%	11	39.3%	16		
	> 10	7	46.7%	9	32.1%	16		
Total		15	100.0 %	28	100.0%	43		

Shorter duration of diabetes mellitus (<5 years) was seen in a greater number of patients in NDKD group when compared to the DKD group. Chronically occurring diabetes mellitus was seen in a higher percentage of people in the DKD group.

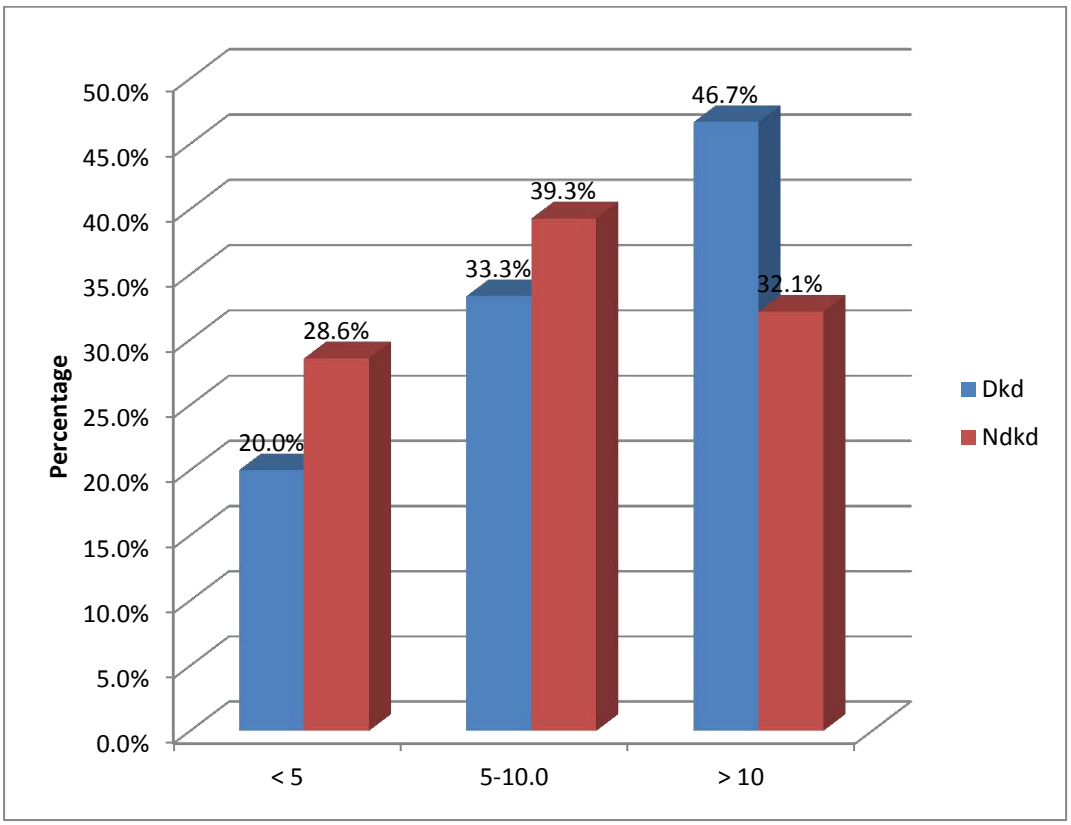


Figure 24: Diabetes duration in NDKD and DKD (n=43)

Table 27: Diabetes control in non-diabetic kidney disease and diabetic kidney disease (n=23)

		DKD		NDKD		Total	Chi-square value	p-value
Diabetes Mellitus On regular treatment	No	4	21.1%	7	29.2%	11	0.811	0.368
	Yes	11	57.9%	21	87.5%	33		
HBA1c	< 8	3	30.0%	6	46.2%	9	0.592	0.442
	> 8	7	70.0%	7	53.8%	14		
Total		10	100.0%	13	100.0%	23		

A total of 23 patients had HBa1C evaluation done. In the DKD group, larger proportion of people had poor glycemic control (HBa1C >8) as compared to the NDKD group where almost an equal proportion of people had fair glycemic control (HBa1C < 8). However, it was not statistically significant

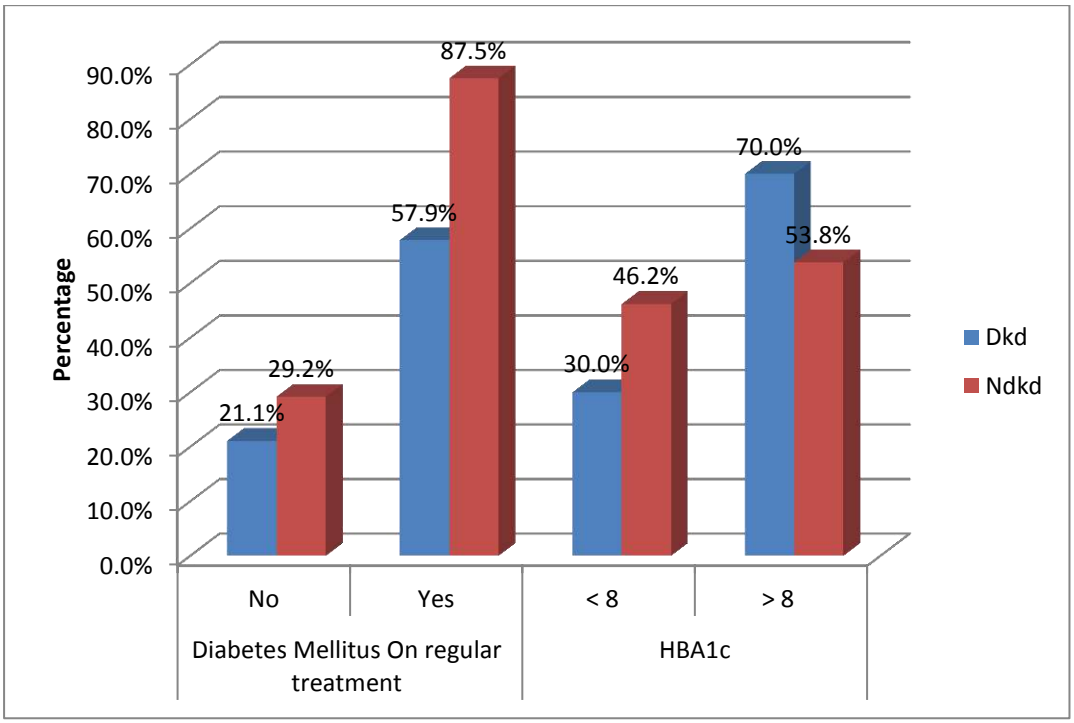


Figure 25: Diabetes control in non-diabetic kidney disease and diabetic kidney disease (n=23)

Table 28: Diabetic retinopathy in non-diabetic kidney disease and diabetic kidney disease (n=43)

		DKD		NDKD		Total	Chi-square value	P-value
Fundus	Non proliferative diabetic retinopathy	7	46.7%	9	32.1%	16	2.452	0.653
	No diabetic retinopathy	8	53.3%	19	67.9%	27		
Total		15	100.0%	28	100.0%	43		

NPDR was seen in 7 patients (46.7%) in DKD group and in 9 patients (32.1%) in the NDKD group had NDPR. Thus, NPDR was more common in the DKD group but was not statistically significant.

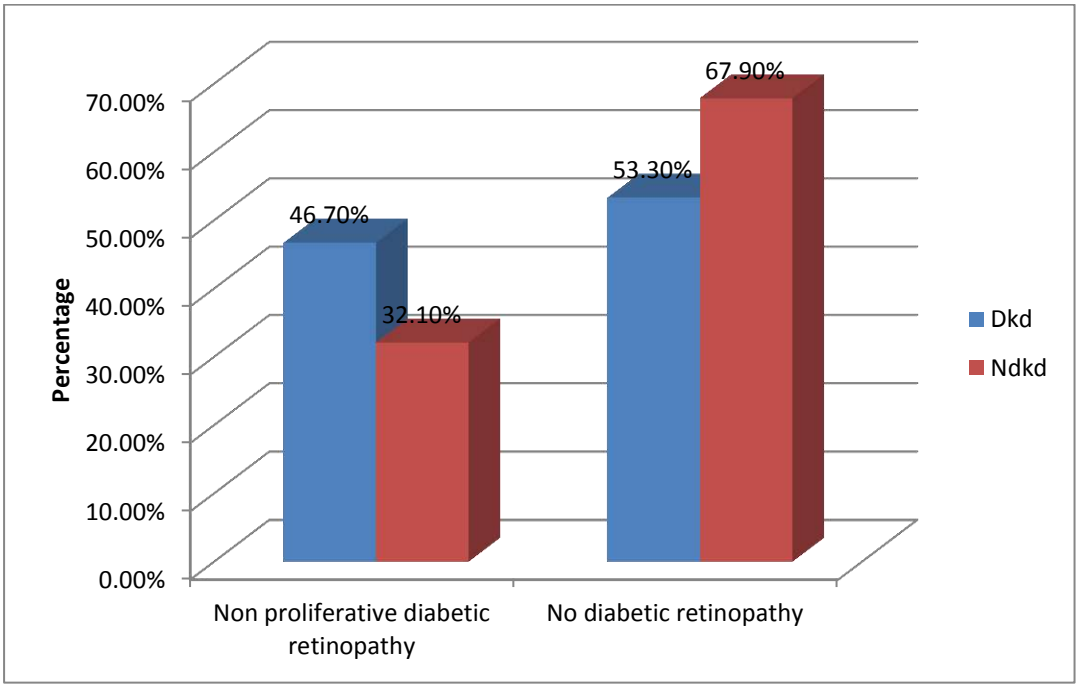


Figure 26: Diabetic retinopathy in non-diabetic kidney disease and diabetic kidney disease (n=43)

Table 29: Hematuria NDKD vs DKD (n=43)

		DKD	%	NDKD	%	Total	Chi-square value	p-value
Microscopic haematuria	< 5	15	100.%	23	82.1%	38	3.031	0.145
	> 5	0	0.0%	5	17.9%	5		
Total		15	100.%	28	100.0%	43		

Significant hematuria was seen only in 5 out of 28 patients (17.9%) in the NDKD group.

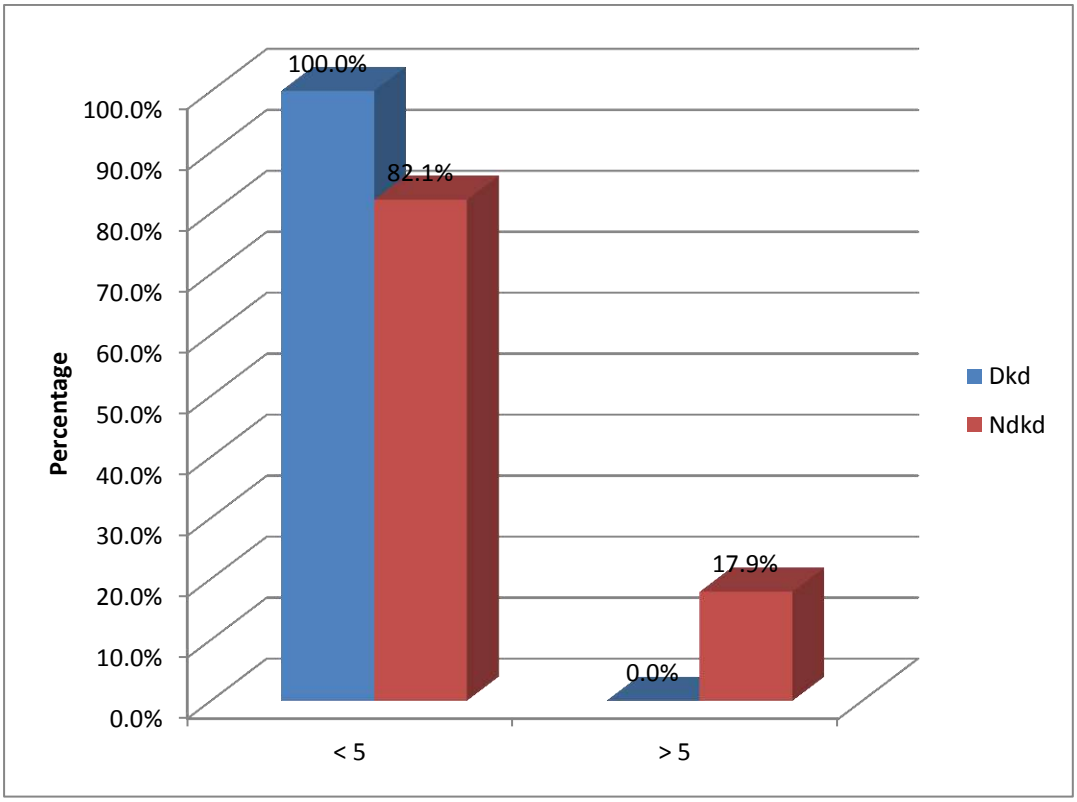


Figure 27: Hematuria NDKD vs DKD (n=43)

Table 30: UPCR in diabetic kidney disease vs non diabetic kidney disease (n=33)

		DKD	%	NDKD	%	Total	Chi-square value	p-value
UPCR	0.2-3.5	2	20%	8	34.7%	10	0.721	0.682
	> 3.5	8	80%	15	65.3%	23		
Total		10	100%	23	100%	33		

15 out of 23 patients (65%) in the NDKD group had nephrotic range proteinuria. 8 out 10 (80%) patients in the DKD group showed nephrotic range proteinuria

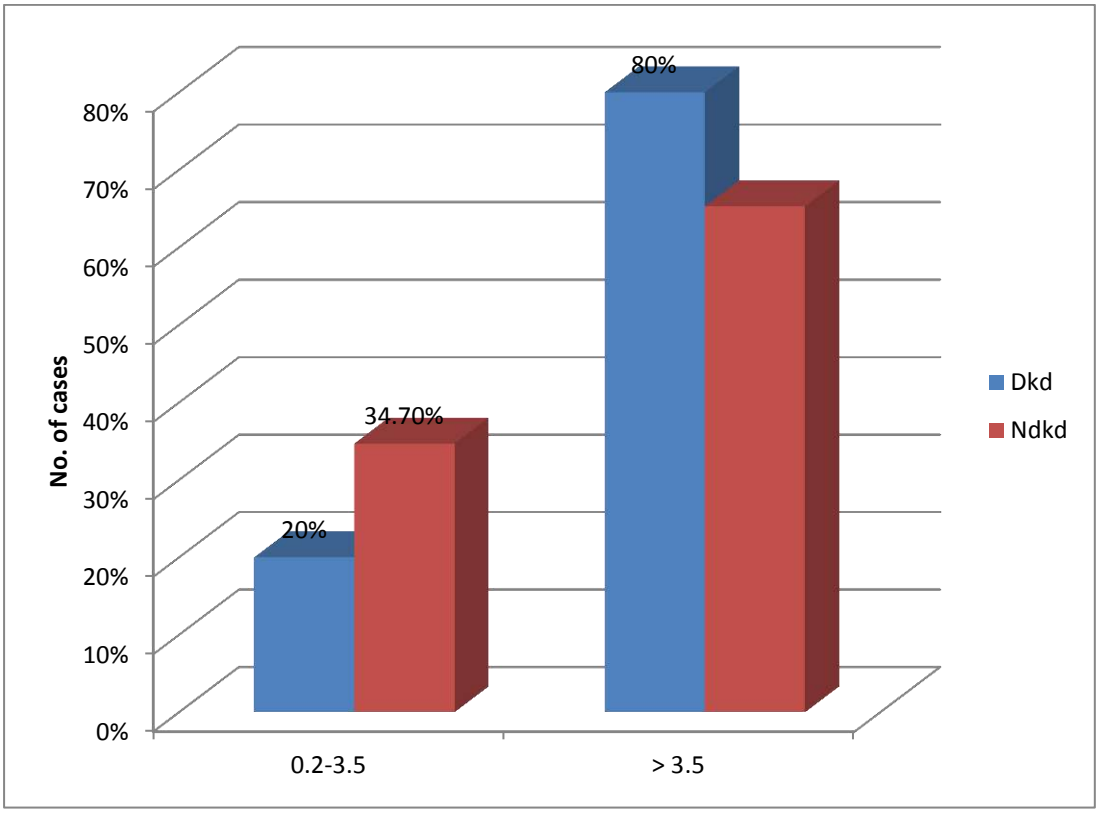


Figure 28: UPCR in diabetic kidney disease vs non diabetic kidney disease (n=33)

Table 31: Autoimmune profile (n=17)

Others:	No. of cases	Percentage
ANA profile positive	1	5.9%
ANA IF positive	4	23.5%
ANCA profile positive	1	5.9%

17 patients had an auto-immune workup done out of which 6 positive profiles were recorded. 4 patients had positive ANA IF, 1 had positive ANA profile and ANCA profile each.

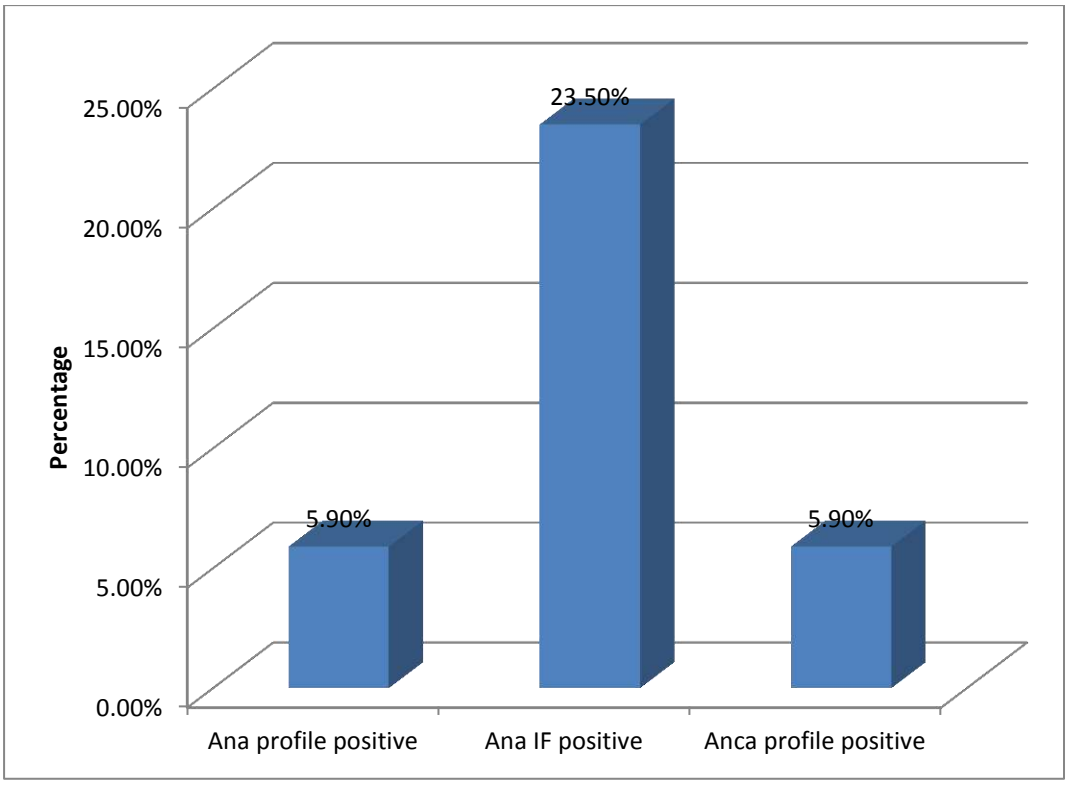


Figure 29: Autoimmune profile (n=17)

Table 32: Autoimmune profiling in non-diabetic kidney disease and diabetic kidney disease (n=6)

	Dkd		Ndkd		Total	Histopathology (NDKD)
ANA profile positive	0	0.0%	1	5.8 %	1	Progressive glomerulonephritis
ANA IF positive	1	5.8%	3	18%	4	I. IgA nephropathy II. Membranous nephropathy III. Immune complex mediated glomerulonephritis (IgA dominated)
ANCA profile positive	0	0.0%	1	5.8%	1	Crescentic glomerulonephritis

ANA IF was positive in 3 patients out of 17. One case of IgA nephropathy, membranous nephropathy and immune complex mediated glomerulonephritis were observed in these patients. In the ANCA profile positive patient, histopathology revealed crescentic glomerulonephritis which was significant. ANA positive profile revealed progressive glomerulonephritis on histopathology.

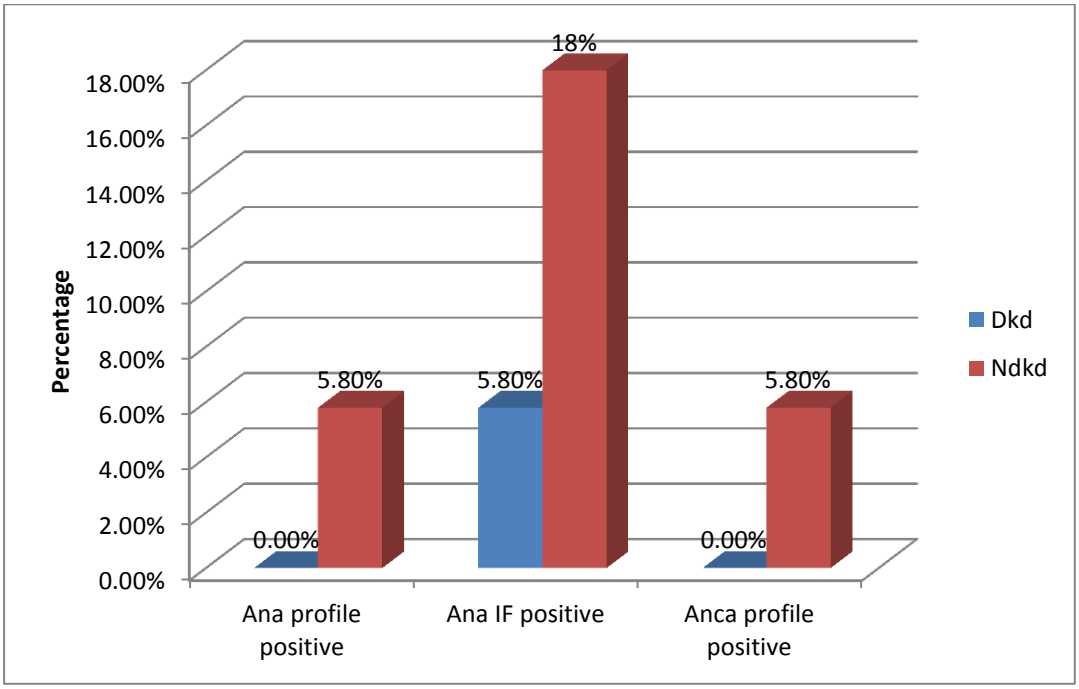


Figure 30: Autoimmune profiling in non-diabetic kidney disease and diabetic kidney disease (n=6)

DISCUSSION

Proteinuria or renal failure in diabetic patients is usually interpreted as a clinical manifestation of diabetic nephropathy. However, not all diabetic subjects presenting with proteinuria or worsening renal failure are due to diabetic nephropathy and can be due to non-diabetic kidney disease. Kidney biopsy though invasive can differentiate diabetic nephropathy from non-diabetic kidney diseases. Some non-diabetic kidney diseases diagnosed on kidney biopsy such as minimal change disease, IgA nephropathy and membranous nephropathy are often treatable and revert the worsening renal failure.

Our study was done in patients with diabetes mellitus who presented with worsening nephropathy. Using pre-defined criteria, patients underwent kidney biopsy with the suspicion that diabetes alone would not be the sole culprit for the worsening, and that non-diabetic kidney disease could be the reason for the same.

The study was conducted in the Department of Medicine and Department of Nephrology in DMC & Hospital, Ludhiana. 43 cases of suspected non-diabetic kidney disease between 1st October 2022 and 31st March 2024 who underwent kidney biopsy were included in the study from both indoor and outdoor departments of the hospital. Clinical and laboratory data of all the 43 patients was pooled together and was analyzed.

Of the 43 patients included in our study, 27 patients (62.8%) were males and 16 patients (37.2%) were females. The male to female ratio was 1.7:1. The male preponderance observed in our study, concurs with the older study by Arora et al [15] and by Fontana et al, [33] both of which have reported male to female ratios of 1.7:1 and 2.6:1 respectively and a study by Grujicic et al [3], a single centre study, stated male to female ratio of 1:1. Similarly in a study done by Sharma et al. [17] male preponderance was seen with male to female ratio of 1.5:1. In studies done by Narayan P et al [23], male to female ratio was 4:1, by Zeng et al [32] male to female ratio

was 2:1 and by He X et al [35] male preponderance was seen with male to female ratio of 2:1.

The mean age in our study was 57.16 ±11.4 years with a range of 31 to 76 years. Arora et al [15] in his study has reported the mean age of 52 ±11.04 years and Fontana et al [33] in his study reported mean age of 62.65 ± 12.35 years with the range of 50 to 74 years. In our study 4 patients (9.3%) were younger than 40 years, 8 patients (18.6%) among 41-50 year age group, 13 patients (30.2%) in 51-60 age group and 12 patients (27.9%) in 61-70 age group. The 5thdecade was the most common age group at presentation in our study corroborated with studies done by Arora et al [15], Grujicic et al [3], Sharma et al [17], and Narayan P et al. [23]

Oedema feet was the most common presenting symptom occurring in 65.1% patients followed by decreased urine output in 39.5 % of patients. Dyspnoea was present in 37.2% patients and facial puffiness was seen in 27.9% patients. Study by Narayan P et al [23] showed similar results where oedematous illness was present in 92.2% patients and oliguria in 38.5 % patients.

8 out of 43 patients (18.7%) were on chronic NSAID/indigenous medication use. On general physical examination, 28 patients had oedema feet followed by pallor in 8 patients. Respiratory examination showed 9 out 43 patients having adventitious sounds in the form of rhonchi and crepitations while majority had a normal examination.

34 patients out of 43 were hypertensive (79.0%). This was comparable with a study done by Narayan P et al [23] where in which 78% patients were hypertensive.

In our study it was found that 15 out of 43 patients (35%) had diabetic kidney disease, and 24 patients (56%) had pure non-diabetic kidney disease and 4 patients (9%) had mixed diabetic and non-diabetic kidney disease. Thus, there were a total of 28 patients (65%) of combined NDKD and NDKD + DKD patients.

Study (n=total patients)	DKD (n)	%	NDKD(n)	%	Mixed renal disease(n) (DKD + NDKD)	%
Our study (n=43)	15	35	24	56	4	9.3
Grujicic et al (n=17)	8	47	6	35.2	3	17.6
Sharma et al (n=140)	59	42	48	34	33	24
Arora P et al (n=44)	11	25	27	61.4	6	13.6
Zeng et al Y et al (n=244)	118	48.36	112	45.9	14	5.74
Eswarappa et al M (n=538)	114	48.3	44	18.6	78	33
Narayan P et al (n=538)	166	33	262	49	110	20
Basu et al et al (n=110)	75	68.4	20	18.2	17	15.4
Erdogmus et al et al (n=48)	20	41.7	24	50	4	8.3

We compared our findings with the other studies done previously as tabulated above. In a study done by Wilfred et al [21] in 2013, they found that 68.8% patients had NDKD with or without concurrent diabetic nephropathy out of the 93 patients who had undergone renal biopsy. Similarly, in a study done by Erdogmus et al[18] had NDKD in 50%, 41.7% in DKD and 8.3% had DKD and NDKD out of the 48 patients they enrolled. Grujicic et al[3] analysed the kidney biopsy findings in 17 diabetic patients out of which 6 had NDKD, 3 had NDKD superimposed on DN and 8 had DN. A study done in 2020 by Sharma M et al [17] Assam enrolled 140 patients. 42% had isolated DN, NDKD seen in 34%, DN and NDKD in 24%. Arora P et al [15] found that out of 44 enrolled patients with clinically suspected NDKD, 61.4% had isolated NDKD, 13.6% had NDKD superimposed on diabetic nephropathy and 25% patients had isolated DN. Zeng Y et al [32] analysed 244 patients and found true DKD in 48.36% patients, NDKD on 45.9% and NDKD superimposed on DKD in 5.74%. Basu et al [6] enrolled 110 subjects and found 68.4% DKD, 18.2% had NDKD and 15.4% had mixed kidney disease. In a study from South India by Eswarappa et al[5] analysed 236 renal biopsies and found features of DKD in 114 patients, NDKD and DN in 78 patients and isolated NDKD in 44 patients. Narayan P et al[23] found DKD in 33% patients, NDKD in 49% patients and NDKD with DKD in 20% patients. Our study results are concurrent with the above studies wherein NDKD ± DKD were more common than DKD. Since our patients underwent kidney biopsy based on pre-set clinical suspicion of NDKD and our results showed more cases of NDKD and mixed renal disease thus emphasizing the need to do kidney biopsy in patients fulfilling the preset suspicion criteria.

However, in some of the studies DKD was more common than NDKD ± DKD. Kanodia V et al [19] analysed kidney biopsy findings done in type 2 DM patients in 2017 and found isolated NDKD in 23%, NDKD superimposed on underlying DN in 23% and 54% had isolated DN. Souza et al [28] in 2020 evaluated 79 kidney biopsies and found 58% patients had DN, 42% had NDKD. Mohammad et al[34] analysed 82 diabetes mellitus

patients who underwent kidney biopsy found DKD in 57.3% while rest of patients had NDKD.

The most common pathology in NDKD was AIN in 5 patients (16.7%) followed by IgA nephropathy, Crescentic glomerulonephritis and Focal proliferative glomerulonephritis accounting for 3 patients each (7%). Diabetic nephropathy was seen in 15 patients (35.0%) and 4 patients had mixed renal disease (9.3%). A meta-analysis by Fiorentina[36] were similar to our findings of the increasing prevalence of acute tubulo-interstitial nephritis. Study done by Prasad N[23] found the most common NDKD pathology as AIN in 33.8% patients followed by IRGN in 13.9% patients. Similarly, Mahesh E[5] showed AIN as the most frequent NDKD pathological diagnosis, followed by post-infectious glomerulo-nephritis and membranous nephropathy. Identical results were seen in a study done by Soni et al. [26]

In the other studies, the most common cause of NDKD were diagnoses other than AIN. Research done by Sharma SG et al [25] showed FSGS as the most common pathology, followed by hypertensive nephrosclerosis, ATN and IgA nephropathy. Wilfred et al [21] found chronic interstitial nephritis as the most common lesion in NDKD with or without concurrent DN followed by acute interstitial nephritis. In a study by Erdogmus et al[18] the most common NDKD diagnoses were membranous nephropathy (29.2%), tubulo-interstitial nephritis (20.8%) and IgA nephropathy (12.5%). Sharma M et al [17] found no patients of acute interstitial nephritis, ATN and chronic tubular-interstitial nephritis in patients of isolated NDKD and reported IgA nephropathy to be the most common cause in the Asian population Souza et al [28] found membranous GN (30.3%) and IgA nephropathy (24.4%) as the most prevalent NDKDs. Arora et al[15] found minimal change disease (19.2%) as the most common NDKD followed by lupus nephritis (11.5%).

The increased proportion of AIN as a cause for NDKD, compared to other studies may be explained by the increased use of OTC (over-the-counter) pain killers and antibiotics by our group of patients. An increasing incidence of AIN attributable to drug use has been documented in other studies as well. In a retrospective analysis of community-acquired acute kidney injury (AKI), reported from an advanced care centre in north India, AIN was found

to be the usual routine cause of AKI in patients who underwent renal biopsy. Proton pump inhibitors, commonly available in our population as an over-the-counter drug have also been speculated as a causative agent of AIN.

4 patients had mixed renal disease were present in our study. Diabetic nephropathy with features of acute interstitial nephritis accounted for 2 cases followed by DN with pyelonephritis and DN with FSGS and AIN in 1 each. Sharma M et al [17] showed similar results in mixed renal disease wherein DN + CTIN was the most common finding followed by DN + AIN followed by DN + IgA nephropathy. Arora et al[15] showed DN with pyelonephritis as most frequent, followed by DN with ATN, DN with thrombotic microangiopathy and DN with lupus nephritis. Sharma et al[17] found DN with ATN as the most common cause followed by FSGS with DN and hypertensive nephrosclerosis. Narayan P[23] and colleagues found ATIN as the most common mixed pathology followed by post infectious glomerulo-nephritis.

Non-diabetic kidney disease was most frequently diagnosed in the 51–60 and 61-70 age group, whereas age group 51-60 was most common for diabetic kidney disease followed by 61–70-year age group as the second commonest.

NDKD was more common in the 41-50 years age group as compared to the DKD group. (21.4% vs 13.3%). The mean age in non-diabetic kidney disease was 55.86 $\pm$ 11.29 which was less than the mean age in diabetic kidney disease was 59.60 $\pm$ 11.42 though it was not statistically significant.

Both diabetic kidney disease and non-diabetic kidney disease were more common in males representing 60.0% and 64.3% respectively. Male to female ratio for DKD was 1:1.5 vs 1:1.8 in the NDKD group.

Majority of patients in both groups had oedema of the feet as a major presenting symptom. Facial puffiness was more common in NDKD in comparison to the DKD group. 9 patients in the NDKD group had dyspnea as a presenting symptom vs 7 patients in the DKD group. 5 patients had

decreased urine output in the DKD group vs 12 patients in the NDKD group.

8 out of 43 patients (18.7%) had history of chronic NSAID /indigenous medication. 2 patients in the DKD group and 6 patients in the NDKD group were found using chronic NSAID/indigenous medicine. Attempts to find correlation between drug intake and NDKD were done. NDKD histopathological analysis showed 4 cases of acute interstitial nephritis out of the 6 patients taking these drugs in this group. Thus, nephrotoxic medications and herbal remedies exposure may also contribute to acute tubulo-interstitial nephritis in a pre-existent state of diabetes-related vascular ischemia. 2 patients in the DKD group were exposed to the same variable with one case showing progressive glomerulo-nephritis.

The mean number of years of diabetes in patients with non-diabetic kidney disease group (9.91 +/- 6.40) was less in comparison in to diabetic nephropathy group (11.13 +/- 6.40) but was not statistically significant. Shorter duration of diabetes has been shown to be predictor of underlying non-diabetic kidney disease. Duration of diabetes mellitus was shorter in patients with NDKD in studies done by Sharma SG et al[25], Wilfred et al[21], Kanodia V et al [19], Erdogmus et al [18] and Zeng et al. [32] Souza DA et al[28] found that variable of diagnosis of DM of 5 years or less was significantly higher in the isolated NDKD group as compared to NDKD/DKD and DKD group. In a study done by Arora P[15] duration of DM was significantly shorter in isolated NDKD group as compared to NDKD/NDKD and DKD group as well.

Shorter duration of diabetes mellitus (<5 years) was seen in a greater number of patients in NDKD group when compared to the DKD group (28.6% vs 20.0%). Diabetes mellitus of more than 10 years was seen in a higher percentage of people in the DKD group (46.7% vs 32.1%). Souza DA et al [28] in their study observed duration of diabetes less than 5 years in 57.58% of the NDKD plus NDKD/DKD group and 17.39% in the DKD group. Also, they observed diabetes duration of more than 10 years in 41.3% of the DKD group vs 21.21% in the NDKD plus NDKD/DKD group as in our study. Similar results were observed by Zeng Y et al[32], Prasad N et al [23] and Singh S et al. [29]

Number of patients with hypertension were more in the DKD group (93.3%) vs NDKD group (71.4%). It was however not statistically significant. Duration of hypertension was significantly more (p= 0.026) in the diabetic kidney disease group than in the non-diabetic kidney disease group in this study. These findings concur with study done by Wilfred et al[21], Kanodia V et al[19], Erdogmus et al[18], and Sharma M et al.[17] High prevalence of hypertension in the diabetic nephropathy group can be explained by the fact that hypertension itself is responsible for progression of diabetic nephropathy.

We found diabetic retinopathy in 39.5% of patients suggests a declining correlation between the two micro-vasculopathies, similarly evidenced by Pedro et al., [37] who found diabetic retinopathy in less than 30% patients. Narayan P et al [23] found diabetic retinopathy in 29.7% patients.

Our study showed that 32.1% of patients had NPDR and 67.9% patients did not have diabetic retinopathy in the NDKD group whereas 46.7% patients had NPDR while 53.3% of patients did not have of diabetic retinopathy in the DKD group. This is also evidenced by Narayan P et al [23] who found diabetic retinopathy in 45.8% in the DKD group and 21.3% in the NDKD group. A study by Sethi J et al [24] found that 46% of patients with diabetic nephropathy and 26% patients of pure non-diabetic kidney disease had diabetic retinopathy. Similar findings were seen in research done by Basu et al, [6] where 95% patients of NDKD group had absence of diabetic retinopathy and 30% patients of DKD did not have diabetic retinopathy. Arora P[15] found an equal distribution of diabetic retinopathy amongst diabetic nephropathy and non-diabetic kidney disease. Studies by S. Singh[29], Zeng et al Y[32], Wilfred D[21], and Sharma M et al[17] had similar comparisons between the two groups.

Thus, the absence of diabetic retinopathy may point towards non-diabetic kidney disease. However, the presence of diabetic retinopathy does not forecast the presence of diabetic kidney disease, and that effort should be taken to exclude non diabetic kidney disease wherever suspected as also evidenced by previous studies.

Microscopic haematuria was seen more in non-diabetic kidney disease as compared to diabetic kidney disease. This finding aligned with studies done

by Basu et al, [6] Eswarappa et al[5], Zeng et al[32], Wilfred et al[21], Erdogmus et al[18], Sharma M et al[17], and Souza et al. [28]

UPCR was analysed in 33 patients. Greater number of patients in the DKD group (80%) as compared to NDKD group (65%) presented with a UPCR >3.5 indicating nephrotic range proteinuria. Studies done by Souza et al[28], Sharma et al[17], Erdogmus et al[18], Kanodia V et al[19], Sharma SG et al[25], Arora et al[15], Zeng et al[32], and Singh et al[29] corroborated our findings. Non-nephrotic range of UPCR was seen commonly in diabetic nephropathy and mixed renal disease.

A subset of 17 patients had an autoimmune workup done. ANA IF was positive in 4 individuals. In ANA IF positive individuals under NDKD group, IgA nephropathy, immune complex dominated GN (IgA dominated), and membranous nephropathy were seen on histopathology. ANA IF was positive in one patient under DKD group and showed just diabetic nephropathy. The patient whose ANA profile was positive under the NDKD group showed progressive glomerulonephritis. ANCA profile was positive in one individual in the NDKD group and histopathology showed crescentic glomerulonephritis. A study by Grujicic et al[3] showed ANA IF positivity with membranous glomerulonephritis on biopsy and ANCA positive profile showed crescentic GN. Similar results were seen in a study by Narayan Prasad et al. [23]

To conclude, the changing pathology of non-diabetic kidney disease in our study could be partially attributed to epidemiological changes, history of drug intake (NSAIDS, indigenous medicine) and increased infections in our patients.

As diabetes epidemic rises, there is an increasing need for internists and nephrologists to remain vigilant and weigh the risks and benefits of renal biopsy in diabetic patients. Routinely a renal biopsy is not indicated in patients with diabetes, it is only done in diabetic patients presenting with atypical features such as microscopic haematuria, sudden rise in creatinine, sudden onset nephrotic syndrome, absence of diabetic retinopathy, and duration of diabetes < 5 years. The precise diagnosis of various NDRDs has obvious therapeutic and prognostic implications as

many NDRDs (e.g., minimal change disease, membranous nephropathy, IgA nephropathy) are often treatable and even curable with appropriate management. Even when a coexistent NDRD does not have a specific treatment, the information obtained by uncovering the histopathologic lesion by kidney biopsy helps in better prognostication of the worsening renal failure in diabetic patients.

SUMMARY AND CONCLUSION

The study was conducted in the Department of Medicine and Department of Nephrology in DMC & Hospital, Ludhiana. 43 cases of suspected non-diabetic kidney disease between 1st October 2022 and 31st March 2024 who underwent kidney biopsy were included in the study from both indoor and outdoor departments of the hospital. Clinical and laboratory data of all the 43 patients was pooled together and was analysed.

- The majority of patients were in the age group 51-60 years (30.2%).
- Overall, a male preponderance was seen with a male to female ratio of 1:1.7.
- Most common symptoms were edema feet in 65.1%, followed by oliguria in 39.5% of the patients.
- Renal biopsy revealed 24 out of 43 patients having pure non-diabetic kidney disease (56.0%). 15 patients (35%) had pure diabetic kidney disease and 4 patients (9.3%) had a mixed picture of non-diabetic kidney disease plus diabetic kidney disease.
- Acute interstitial nephritis (12%) was the most common form of non-diabetic kidney disease followed by IgA nephropathy, crescentic glomerulonephritis and focal proliferative glomerulonephritis (7.0% each). In mixed renal disease, the most common pathology seen was diabetic nephropathy with acute tubulointerstitial nephritis.
- NDKD was more common in the 41-50 years age group as compared to the DKD group (21.4% vs 13.3%).
- The mean age in non-diabetic kidney disease was 55.86 $\pm$ 11.29 which was less than the mean age in diabetic kidney disease was 59.60 $\pm$ 11.42 though it was not statistically significant.
- A total of 8 patients were taking NSAID/Indigenous medication among our study group out of which 6 were in the NDKD group while 2 were in the DKD group. AIN and DN with AIN were the most common pathologies in the NDKD group taking these medications.

- Greater number of patients with diabetic kidney disease were hypertensive as compared to non-diabetic kidney disease group.
- Hypertension duration in non-diabetic kidney disease group was 4.98 ± 2.86 years and in the diabetic kidney disease group it was 8.07± 4.65 years which was statistically significant.
- Shorter duration of diabetes mellitus (<5 years) was seen in a greater number of patients in NDKD group when compared to the DKD group. (not significant). Diabetes mellitus duration of more than 10 years was seen in higher percentage of people in the DKD group.
- The diabetic kidney disease group had more patients with non-proliferative diabetic retinopathy as compared to the NDKD group (46.7% vs 32.1%).
- Significant hematuria was seen in 5 out of 28 patients (17.9%) in the NDKD group while none of the patients in the DKD group had hematuria.
- UPCR was done in 33 patients out of which 70% showed nephrotic range proteinuria, 65% in the NDKD group and 80% in the DKD group.
- 17 patients had an autoimmune workup done. 3 patients were ANA IF positive which had IgA nephropathy, membranous nephropathy and immune complex mediated glomerulonephritis. 1 patient had ANCA profile positive revealing crescentic glomerulonephritis.

To conclude, our study showed NDKD in 56% of the patients and mixed renal disease in 9.3% patients. AIN was the most common histopathological diagnosis in NDKD patients. Statistically significant shorter duration of hypertension was found in the NDKD group. Non statistically significant differences were found in the younger age group (41-50 years), lesser mean age (55.86 ± 11.29 years) and lesser duration of diabetes in the NDKD group. Drug intake led to acute interstitial nephritis in 4 patients. NPDR was seen more in DKD when compared to NDKD but was not statistically significant. Microscopic hematuria was seen only in 17.9% of the NDKD group of patients.

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S.No	Name	MRD number	Age	AGE GROUP	Sex	Occupation	Date of admission	History of present illness_Dyspnoea (Yes/No)	Chest pain (Yes/No)	Decreased urine output (Yes/No)	Facial puffiness (Yes/No)	Oedema feet (Yes/No)	Haematuria (Yes/No)	Any other symptoms:	Drug intake (NSAIDS, Antibiotics, Indigenous medications etc) (Yes/No)
1	US	1520607	58	3	FEMALE	HOUSEWIFE	01-12-2023	NO	NO	NO	NO	YES	NO	FROTHURIA	NO
2	ML	1630041	63	4	MALE	RETIRED	01-11-2023	YES	NO	NO	NO	NO	YES	NO	NO
3	LK	1623059	70	4	FEMALE	HOUSEWIFE	01-02-2024	YES	NO	NO	NO	NO	NO	COUGH	NO
4	C	1583324	58	3	MALE	RETIRED	05-09-2023	NO	NO	NO	NO	YES	NO	NO	NO
5	SR	1525172	52	3	FEMALE	HOUSEWIFE	31-05-2023	YES	NO	YES	NO	NO	NO	NO	NO
6	MK	1649736	49	2	FEMALE	HOUSEWIFE	18-01-2024	YES	NO	NO	NO	YES	NO	COUGH	YES (NSAIDS)
7	OS	1276734	72	5	MALE	RETIRED	18-11-2023	YES	NO	NO	NO	YES	NO	VOMITING/NAUSEA	NO
8	SK	1645293	50	2	MALE	BUISNESS	06-03-2024	NO	NO	NO	NO	YES	NO	DECREASED APPETITE, GENERALISED WEAKNESS	NO
9	RC	1550289	66	4	MALE	RETIRED	14-08-2023	YES	NO	YES	NO	YES	NO	COUGH	NO
10	VP	1294462	71	5	FEMALE	HOUSEWIFE	11-09-2023	YES	NO	YES	YES	YES	NO	CONSTIPATION	NO
11	JK	1469904	42	2	MALE	BUISNESS	28-08-2023	YES	NO	YES	NO	YES	NO	NO	NO
12	AM	1567711	53	3	FEMALE	HOUSEWIFE	26-08-2023	NO	NO	NO	NO	NO	YES	FEVER	NO
13	JS	1571870	55	3	MALE	BUISNESS	16-08-2023	NO	NO	NO	NO	YES	NO	BODY ACHES	NO
14	AK	1497806	64	4	MALE	BUISNESS	18-07-2023	NO	NO	NO	NO	YES	NO	FROTHURIA	YES (NSAIDS)
15	PS	1115147	76	5	MALE	RETIRED	12-08-2023	NO	NO	YES	NO	NO	NO	COUGH	NO
16	BS	900377	43	2	MALE	BUISNESS	11-07-2023	NO	NO	NO	NO	YES	NO	ABDOMINAL DISTENSION	NO
17	MAB	1563716	63	4	MALE	RETIRED	01-08-2023	NO	NO	YES	NO	NO	NO	NO	NO

S.No	Diabetes Mellitus Number of years:	Diabetes Mellitus On regular treatment (Yes/No)	Hypertension Number of years:	Hypertension On regular treatment (Yes/No)	Any other relevant past history:	Smoking (Yes/No)	Alcohol (Yes/No)	Any other drug abuse (Yes/No)	BMI (kg/m2)	Pulse rate (x/min)	SBP	DBP	Respiratory rate:	Cyanosis:	Clubbing:	Icterus:	Pallor:	JVP:
1	20	YES	6 YEARS	YES	NO	NO	NO	NO	21	72	110	90	20	NO	NO	NO	NO	NO
2	10	YES	NO	NO	NO	NO	NO	NO	19	80	110	90	18	NO	NO	NO	NO	NO
3	18	YES	NO	NO	NO	NO	NO	NO	24	75	110	70	18	NO	NO	NO	NO	NO
4	12	YES	NO	NO	NO	NO	NO	NO	24	70	110	70	18	NO	NO	NO	NO	NO
5	10	YES	NO	NO	NO	NO	NO	NO	22	90	120	70	18	NO	NO	NO	NO	NO
6	25	YES	3 YEARS	YES	NO	NO	NO	NO	27	85	110	70	22	NO	NO	NO	NO	NO
7	10	YES	15 YEARS	YES	NO	YES	NO	NO	26	80	110	90	20	NO	NO	NO	NO	NO
8	3.5	YES	3.5 YEARS	NO	NO	NO	NO	NO	21	80	110	90	18	NO	NO	NO	NO	NO
9	20	YES	1 YEAR	YES	NO	NO	NO	NO	26	75	110	70	22	NO	NO	NO	NO	NO
10	20	YES	10 YEARS	YES	NO	NO	NO	NO	22	85	140	90	21	NO	NO	NO	NO	NO
11	10	YES	YES	YES	YES	NO	NO	NO	24	80	110	90	18	NO	NO	NO	NO	NO
12	2	YES	NO	NO	NO	NO	NO	NO	28	90	120	70	18	NO	NO	NO	NO	NO
13	10	YES	10 YEARS	YES	NO	NO	NO	NO	26	80	110	70	18	NO	NO	NO	NO	NO
14	2	YES	2 MONTHS	YES	NO	NO	NO	NO	27	85	120	70	18	NO	NO	NO	NO	NO
15	10	YES	8 YEARS	YES	NO	NO	NO	NO	24	80	110	70	18	NO	NO	NO	NO	NO
16	15	YES	2 YEARS	YES	NO	NO	NO	NO	21	80	110	90	18	NO	NO	NO	NO	NO
17	22	YES	10 YEARS	YES	NO	NO	NO	NO	24	72	140	90	16	NO	NO	NO	NO	NO

S.No	Respiratory exam:	Abdominal exam	Cardiovascular exam	Central nervous system exam, Reflexe	Plantar	Vibration sense	Fundus	HB	PCV	Plateletcount	ESR	TLC	DLC	Blood urea	S. creatinine	UPCR
1	NVBS. NO ADDED SOUNDS	SOFT, NON-TENDER. NO ORGANOMEGALY	S1 AND S2 PRESENT. NO ADDED SOUNDS	DTR PRESENT	B/L DOWN	PRESENT	MILD NPDR	9.5	29.5	170	61	8.4	N - 69%, L- 17.4 %, M - 12.8 %, E- 0.4%, B- 0.4%	115	8.36	5.6
2	NVBS. NO ADDED SOUNDS	SOFT, NON-TENDER. NO ORGANOMEGALY	S1 AND S2 PRESENT. NO ADDED SOUNDS	DTR PRESENT	B/L DOWN	PRESENT	NO DIABETIC RETINOPATHY	9.1	27.3	130	67	6.2	N - 66.5%, L- 24.4 %, M - 7.9 %, E- 0.8%, B- 0.4%	82	2.92	6
3	B/L CREPITATIONS HEARD. NO ADDED SOUNDS	SOFT, NON-TENDER. NO ORGANOMEGALY	S1 AND S2 PRESENT. NO ADDED SOUNDS	DTR PRESENT	B/L DOWN	PRESENT	MILD NPDR	7.3	23.8	274	60	5.14	N - 71.1%, L- 18.4 %, M - 9.3%, E- 1.0%, B- 0.4%	91	6.43	4
4	B/L CREPITATIONS HEARD. NO ADDED SOUNDS	SOFT, NON-TENDER. NO ORGANOMEGALY	S1 AND S2 PRESENT. NO ADDED SOUNDS	DTR PRESENT	B/L DOWN	PRESENT	NO DIABETIC RETINOPATHY	11.6	34.9	226	40	6.3	N - 62.7%, L- 20.9%, M - 14% %, E- 2.3%, B- 0.4%	129	2.66	0.4
5	B/L CREPITATIONS HEARD. NO ADDED SOUNDS	SOFT, NON-TENDER. NO ORGANOMEGALY	S1 AND S2 PRESENT. NO ADDED SOUNDS	DTR PRESENT	B/L DOWN	PRESENT	NO DIABETIC RETINOPATHY	9	28.4	310	55	10.75	N - 73%, L- 17.2%, M - 3.9% %, E- 5.5%, B- 0.4%	268	10.79	7
6	B/L CREPITATIONS HEARD. NO ADDED SOUNDS	SOFT, NON-TENDER. NO ORGANOMEGALY	S1 AND S2 PRESENT. NO ADDED SOUNDS	DTR PRESENT	B/L DOWN	PRESENT	MILD NPDR	7.3	22.6	205	44	7.6	N - 77.1%, L- 19.9%, M - 11% %, E- 0.3%, B- 0.4%	140	8.19	13.4
7	NVBS. NO ADDED SOUNDS	SOFT, NON-TENDER. NO ORGANOMEGALY	S1 AND S2 PRESENT. NO ADDED SOUNDS	DTR PRESENT	B/L DOWN	PRESENT	SEVERE NPDR	7.7	23.5	106	36	7.4	N - 66.5%, L- 24.4 %, M - 7.9 %, E- 0.8%, B- 0.4%	116	4.54	7
8	NVBS. NO ADDED SOUNDS	SOFT, NON-TENDER. NO ORGANOMEGALY	S1 AND S2 PRESENT. NO ADDED SOUNDS	DTR PRESENT	B/L DOWN	PRESENT	MILD NPDR	9.5	28.7	248	28	7.4	N - 73%, L- 17.2%, M - 3.9% %, E- 5.5%, B- 0.4%	183	7.2	8
9	B/L CREPITATIONS HEARD. NO ADDED SOUNDS	SOFT, NON-TENDER. NO ORGANOMEGALY	S1 AND S2 PRESENT. NO ADDED SOUNDS	DTR PRESENT	B/L DOWN	PRESENT	SEVERE NPDR	5.5	17.2	205	40	4.9	N - 62.7%, L- 20.9%, M - 14% %, E- 2.3%, B- 0.4%	112	6.19	9
10	B/L CREPITATIONS HEARD. NO ADDED SOUNDS	SOFT, NON-TENDER. NO ORGANOMEGALY	S1 AND S2 PRESENT. NO ADDED SOUNDS	DTR PRESENT	B/L DOWN	PRESENT	NO DIABETIC RETINOPATHY	8.7	28.2	130	36	8.9	N - 69%, L- 17.4 %, M - 12.8 %, E- 0.4%, B- 0.4%	66	1.93	45
11	NVBS. NO ADDED SOUNDS	SOFT, NON-TENDER. NO ORGANOMEGALY	S1 AND S2 PRESENT. NO ADDED SOUNDS	DTR PRESENT	B/L DOWN	PRESENT	MILD NPDR	10.4	34.6	150	38	12.46	N - 71.1%, L- 18.4 %, M - 9.3%, E- 1.0%, B- 0.4%	42	0.64	7.7
12	B/L RHONCHI HEARD.	SOFT, NON-TENDER. NO ORGANOMEGALY	S1 AND S2 PRESENT. NO ADDED SOUNDS	DTR PRESENT	B/L DOWN	PRESENT	NO DIABETIC RETINOPATHY	9	28.6	191	28	10.75	N - 62.7%, L- 20.9%, M - 14% %, E- 2.3%, B- 0.4%	50	1.22	22
13	NVBS. NO ADDED SOUNDS	SOFT, NON-TENDER. NO ORGANOMEGALY	S1 AND S2 PRESENT. NO ADDED SOUNDS	DTR PRESENT	B/L DOWN	PRESENT	SEVERE NPDR	9.5	29.9	319	63	8.4	N - 69%, L- 17.4 %, M - 12.8 %, E- 0.4%, B- 0.4%	183	8.48	86
14	NVBS. NO ADDED SOUNDS	SOFT, NON-TENDER. NO ORGANOMEGALY	S1 AND S2 PRESENT. NO ADDED SOUNDS	DTR PRESENT	B/L DOWN	PRESENT	NO DIABETIC RETINOPATHY	11.4	33.8	154		5.14	N - 71.1%, L- 18.4 %, M - 9.3%, E- 1.0%, B- 0.4%	90	1.08	7.59
15	NVBS. NO ADDED SOUNDS	SOFT, NON-TENDER. NO ORGANOMEGALY	S1 AND S2 PRESENT. NO ADDED SOUNDS	DTR PRESENT	B/L DOWN	PRESENT	NO DIABETIC RETINOPATHY	7.3	22.9	102	40	14.2	N - 62.7%, L- 20.9%, M - 14% %, E- 2.3%, B- 0.4%	100	6.7	10
16	NVBS. NO ADDED SOUNDS	SOFT, NON-TENDER. NO ORGANOMEGALY	S1 AND S2 PRESENT. NO ADDED SOUNDS	DTR PRESENT	B/L DOWN	PRESENT	MILD NPDR	9.1	23.3	150	40	10.75	N - 66.5%, L- 24.4 %, M - 7.9 %, E- 0.8%, B- 0.4%	72	5.04	11.2
17	NVBS. NO ADDED SOUNDS	SOFT, NON-TENDER. NO ORGANOMEGALY	S1 AND S2 PRESENT. NO ADDED SOUNDS	DTR PRESENT	B/L DOWN	PRESENT	MILD NPDR	11.1	35.6	191	44	10.75	N - 77.1%, L- 19.9%, M - 11% %, E- 0.3%, B- 0.4%	207	6.9	7.72

S.No	URINE ROUTINE PROTEIN	URINE ROUTINE GLUCOSE	URINE ROUTINE PUS CELLS	URINE ROUTINE RBC	Sodium	Potassium	Chloride	Total cholesterol	TG	LDL	SGOT	SGPT	ALKALINE PHOSPHATASE	TOTAL BILIRUBIN	DIRECT BILIRUBIN	ALBUMIN	TOTAL PROTEIN	Calcium	Phosphorus
1	3+	2+	0	0	136	4.3	99	260			12	16	45	0.2	0.3	3.4	4.6	8.26	4
2	4+	1+	0	0	143	4.21	115	216	200		14	11	96	0.55	0.1	3.1	4.2	9.1	
3	3+	2+	0	0	138	4.98	100	140	233	40	7	11	129	0.4	0.26	3.71	4.6	8.6	6.4
4	1+	2+	0	2	136	4.3	80				12	11	66	0.6	0.1	3.1	4.2	9.8	3.4
5	3+	1+	2	3	129	4.59	97	222	200	90	9	20	55	0.19	0.07	3.71	4.3	6.9	8.3
6	3+	0	3	2	138	4.21	110	231			5	21	80	0.9	0.3	3.4	3.2	7.8	9
7	3+	2+	0	4	143	3.27	107				49	35	50	0.6	0.1	3	3.8	9.8	8.9
8	2+	2+	0	0	136	4.59	97				12	35	45	0.02	0.03	2.89	6.2	9.1	7.4
9	2+	3+	1	0	139	5.52	109	1.22			14	14	96	0.55	0.1	3.1	4.2	7.7	5.12
10	2+	2+	0	0	141	141	4.95				5	21	80	0.9	0.3	3.4	3.2	8	6
11	2+	3+	0	0	136	3.3	97	260	200	40	20	24	61	0.34	0.57	3.1	4.5	9.4	
12	3+	4+	0	0	139	3.3	107				10	14	66	0.2	0.1	3.71	3.8	8	4.8
13	4+	2+	0	0	136	4.3	100				15	16	45	0.2	0.3	3.4	3	8.43	7.1
14	4+	1+	0	0	139	4.21	109				12	11	90	0.1	0.5	3.1	2.68	9.8	5.12
15	3+	3+	0	3	136	3.3	97				14	12	96	0.55	0.21	3.1	4.2		
16	3+	2+	0	4	138	4.59	107	113	160		49	35	50	0.6	0.1	3	3.8		
17	3+	2+	0	3	136	4.59	97				12	32	88	0.2	0.3	3.4	4		

S.No	Uric acid	Viral markers	HBA1c	USG abdomen	Others:	Histopathology report: Immuno-florescence (if needed)	Histopathology Impression:	NDKD	Final diagnosis	Outcome:	Treatment plan	Treatment started - Yes/No
1	4.5	NON-REACTIVE	NOT DONE	BILATERAL RENALL PARENCHYMAL CHANGES	NOT DONE	IgG: PSEUDOLINEAR ACCENTUATION. KAPPA AND LAMBDA: PSEUDOLINEAR ACCENTUATION	DKD	N	ADVANCED DIABETIC NEPHROSCLEOSIS	ON TREATMENT	STRICT GLYCEMIC CONTROL	YES
2		NON-REACTIVE	NOT DONE	NOT DONE	NOT DONE	NOT SIGNIFICANT	NDKD	Y	ACUTE TUBULO-INTERSTITIAL NEPHRITIS	ON TREATMENT	NSAIDS, ANTIBIOTICS	YES
3		NON-REACTIVE	NOT DONE	BILATERAL RENALL PARENCHYMAL CHANGES	ANA AND ANCA PROFILE - NEGATIVE	NOT SIGNIFICANT	DKD	N	DIABETIC NEPHROPATHY	ON TREATMENT	OHAS AND STRICT GLYCEMIC CONTROL	YES
4		NON-REACTIVE	7.3	ACUTE MEDICAL RENAL DISEASE	NOT DONE	IgG DEPOSITS TUBULAR BASEMENT MEMBRANE	NDKD	Y	ACUTE ON CHRONIC TUBULO-INTERSTITIAL NEPHRITIS	ON TREATMENT	STEROIDS	YES
5	6	NON-REACTIVE	9.3	ACUTE MEDICAL RENAL DISEASE	NOT DONE	NOT SIGNIFICANT	NDKD	Y	ACUTE TUBULO-INTERSTITIAL NEPHRITIS	IMPROVED	-	YES
6		NON-REACTIVE	NOT DONE	BILATERAL RENALL PARENCHYMAL CHANGES	ANA NEGATIVE	NOT SIGNIFICANT	DKD	N	DIABETIC NEPHROPATHY	IMPROVED	OHAS AND STRICT GLYCEMIC CONTROL	YES
7	5	NON-REACTIVE	10.6	BILATERAL RENALL PARENCHYMAL CHANGES	NOT DONE	NOT SIGNIFICANT	DKD	N	DIABETIC NEPHROPATHY	IMPROVED	OHAS AND STRICT GLYCEMIC CONTROL	YES
8	5.6	NON-REACTIVE	5.6	BILATERAL RENALL PARENCHYMAL CHANGES	NOT DONE	NOT SIGNIFICANT	NDKD	Y	ACUTE TUBULO-INTERSTITIAL NEPHRITIS	IMPROVED	STEROIDS	YES
9	7.5	NON-REACTIVE	7.4	BILATERAL RENALL PARENCHYMAL CHANGES	ANCA PROFILE NEGATIVE	NOT SIGNIFICANT	DKD	N	DIABETIC KIDNEY DISEASE	IMPROVED	OHAS AND STRICT GLYCEMIC CONTROL	YES
10	6.9	NON-REACTIVE	7.3	NO SIGNIFICANT FINDINGS	ANA POSITIVE ANCA PROFILE NEGATIVE	IGA - MESANGIAL POSITIVITY +3. KAPPA - MESANGIAL POSITIVITY +2. LAMBDA MESANGIAL +1	NDKD	Y	IGA NEPHROPATHY	IMPROVED	STEROIDS AND STRICT GLYCEMIC CONTROL	YES
11		NON-REACTIVE	NOT DONE	NOT DONE	NOT DONE	NOT SIGNIFICANT	NDKD	Y	PODOCYTOPATHY	IMPROVED	STEROIDS	YES
12	4.4	NON-REACTIVE	9.6	NO SIGNIFICANT FINDINGS	ANA NEGATIVE	C3C - POSITIVE IN MESANGIUM. IGA - POSITIVE IN MESANGIUM	NDKD	Y	IGA NEPHROPATHY	IMPROVED	STEROIDS	YES
13		NON-REACTIVE	NOT DONE	ACUTE MEDICAL RENAL DISEASE	NOT DONE	NOT SIGNIFICANT	DKD	N	DIABETIC KIDNEY DISEASE	IMPROVED	STRICT GLYCEMIC CONTROL	YES
14		NON-REACTIVE	7.5	NOT DONE	ANA POSITIVE	GRANULAR POSITIVITY OF IGG +3 AND C3C +2 NOTED ALONG THE GLOMERULAR CAPILLARY LOOPS	NDKD	Y	MEMRANOUS NEPRHOPATHY	IMPROVED	STEROIDS	YES
15		NON-REACTIVE	NOT DONE	ACUTE MEDICAL RENAL DISEASE	NOT DONE	NO GLOMERULAR DEPOSITS SEEN.	DKD	N	PROGRESSIVE GLOMERULOSCLEROSIS	IMPROVED	ANT -HTN AND OHAS	YES
16		NON-REACTIVE	9	BILATERAL RENALL PARENCHYMAL CHANGES	NOT DONE	NO GLOMERULAR DEPOSITS SEEN.	NDKD	Y	FOCAL PROLIFERATIVE GLOMERULONEPHRITIS	IMPROVED	STEROIDS	YES
17		NON-REACTIVE	NOT DONE	ACUTE MEDICAL RENAL DISEASE	ANA PROFILE POSITIVE	NO GLOMERULAR DEPOSITS SEEN.	NDKD	Y	PROLIFERATIVE GLOMERULONEPHRITIS	IMPROVED	STEROID PULSE F/B ORAL STEROIDS.	YES

S.No	Name	MRD number	Age	AGE GROUP	Sex	Occupation	Date of admission	History of present illness_Dyspnoea (Yes/No)	Chest pain (Yes/No)	Decreased urine output (Yes/No)	Facial puffiness (Yes/No)	Oedema feet (Yes/No)	Haematuria (Yes/No)	Any other symptoms:	Drug intake (NSAIDS, Antibiotics, Indigenous medications etc) (Yes/No)
18	BDM	1533147	61	4	MALE	RETIRED	11-08-2023	NO	NO	YES	NO	YES	NO	WEIGHT LOSS	NO
19	PA	1507415	37	1	MALE	TEACHER	02-05-2023	NO	NO	YES	YES	YES	NO	NO	NO
20	MS	1515093	55	3	MALE	GOVT EMPLOYEE	01-05-2023	NO	NO	YES	YES	YES	NO	DECREASED APPETITE, GENERALISED WEAKNESS	NO
21	P	1541163	56	3	FEMALE	HOUSEWIFE	23-06-2023	YES	NO	YES	YES	YES	NO	NO	NO
22	TK	1492348	73	5	FEMALE	HOUSEWIFE	03-04-2023	YES	NO	YES	YES	YES	NO	NO	NO
23	JK	1488701	63	4	FEMALE	HOUSEWIFE	28-03-2023	NO	NO	YES	YES	YES	NO	FROTHURIA	YES (NSAIDS)
24	PK	1480582	67	4	MALE	RETIRED	14-03-2023	NO	NO	NO	NO	NO	NO	COUGH	YES
25	PS	1470102	31	1	MALE	STUDENT	10-03-2023	NO	NO	NO	NO	YES	NO	FROTHURIA	NO
26	GK	1471777	40	1	FEMALE	HOUSEWIFE	01-03-2023	NO	NO	NO	YES	NO	NO	DECREASED APPETITE, GENERALISED WEAKNESS	YES(INDIGENOUS MEDICATIONS)
27	SK	1462667	72	5	FEMALE	RETIRED	16-02-2023	NO	NO	NO	NO	NO	NO	FEVER	YES(INDIGENOUS MEDICATIONS), HOMEOPATHY MEDICATIONS
28	KK	1460743	55	3	FEMALE	HOUSEWIFE	10-02-2023	NO	NO	NO	YES	YES	NO	COUGH	NO
29	JS	1458203	57	3	MALE	RETIRED	06-02-2023	YES	NO	YES	NO	YES	NO	NO	NO
30	SD	1452470	65	4	FEMALE	HOUSEWIFE	27-01-2023	YES	NO	YES	NO	NO	NO	DECREASED APPETITE, GENERALISED WEAKNESS	NO
31	PK	266645	60	3	MALE	TRADE	01-09-2022	NO	NO	NO	NO	NO	NO	NO	NO
32	AK	1431450	50	2	MALE	BUISNESS	26-12-2022	YES	NO	NO	YES	YES	NO	FROTHURIA	YES (NSAIDS)
33	NSS	1389344	64	4	MALE	FARMING	20-12-2022	YES	NO	YES	NO	YES	NO	NO	YES (NSAIDS)
34	KS	1429889	43	2	MALE	ELECTRICIAN	13-12-2023	NO	NO	NO	NO	NO	NO	DECREASED APPETITE, GENERALISED WEAKNESS	NO
35	TC	1430837	58	3	MALE	SHOPKEEPER	13-12-2022	YES	NO	NO	NO	YES	NO	NO	NO

S.No	Diabetes Mellitus Number of years:	Diabetes Mellitus On regular treatment (Yes/No)	Hypertension Number of years:	Hypertension On regular treatment (Yes/No)	Any other relevant past history:	Smoking (Yes/No)	Alcohol (Yes/No)	Any other drug abuse (Yes/No)	BMI (kg/m2)	Pulse rate (x/min)	SBP	DBP	Respiratory rate:	Cyanosis:	Clubbing:	Icterus:	Pallor:	JVP:
18	10	YES	5 YEARS	YES	NO	YES	NO	NO	19	75	110	70	18	NO	NO	NO	NO	NO
19	10	YES	NO	NO	NO	NO	NO	NO	26	80	120	70	16	NO	NO	NO	NO	NO
20	10	YES	YES	YES	RHEUMATOID ARTHRITIS	NO	NO	NO	22	80	110	70	16	NO	NO	NO	NO	NO
21	4	YES	3 YEARS	YES	NO	NO	NO	NO	26	72	120	70	18	NO	NO	NO	NO	NO
22	0.416666667	YES	4 YEARS	YES	NO	NO	NO	NO	22	85	110	70	16	NO	NO	NO	NO	NO
23	5	YES	3 YEARS	NO	NO	NO	NO	NO	28	80	110	70	18	NO	NO	NO	NO	NO
24	6	YES	3 YEARS	YES	NO	NO	NO	NO	26	75	150	90	16	NO	NO	NO	NO	NO
25	7	YES	3 YEARS	YES	NO	NO	NO	NO	23	72	120	70	18	NO	NO	NO	NO	NO
26	2	NO	NO	NO	NO	NO	NO	NO	28	85	110	90	16	NO	NO	NO	NO	NO
27	20	YES	2 MONTHS	YES	NO	NO	NO	NO	24	70	120	70	18	NO	NO	NO	NO	NO
28	3	YES	10 YEARS	YES	NO	NO	NO	NO	28	72	110	70	16	NO	NO	NO	NO	NO
29	0.5	YES	6 MONTHS	YES	YES	YES	NO	NO	26	90	130	80	18	NO	NO	NO	NO	NO
30	15	YES	10 YEARS	YES	NO	NO	NO	NO	24	75	130	80	16	NO	NO	NO	NO	NO
31	15	YES	5 YEARS	YES	CAD, POST CABG	NO	NO	NO	23	70	140	90	18	NO	NO	NO	NO	NO
32	5	YES	1 MONTH	YES	RISING CREATININE	NO	NO	NO	24	86	120	70	18	NO	NO	NO	NO	NO
33	11	YES	5 YEARS	YES	NO	NO	NO	NO	26	98	150	90	20	NO	NO	NO	YES	NO
34	5	YES	4 YEARS	YES	NO	NO	NO	NO	27	80	120	70	18	NO	NO	NO	NO	NO
35	10	YES	10 YEARS	YES	NO	NO	NO	NO	24	72	140	90	16	NO	NO	NO	NO	NO

S.No	Respiratory exam:	Abdominal exam	Cardiovascular exam	Central nervous system exam. Reflexe	Plantar	Vibration sense	Fundus	HB	PCV	Plateletcount	ESR	TLC	DLC	Blood urea	S. creatinine	UPCR
18	B/L CREPITATIONS HEARD. NO ADDED SOUNDS	SOFT, NON-TENDER. NO ORGANOMEGALY	S1 AND S2 PRESENT. NO ADDED SOUNDS	DTR PRESENT	B/L DOWN	PRESENT	NO DIABETIC RETINOPATHY	12.6	32	170	40	10.75	N - 69%, L- 17.4 %, M - 12.8 %, E- 0.4%, B- 0.4%	180	4.6	4
19	NVBS. NO ADDED SOUNDS	SOFT, NON-TENDER. NO ORGANOMEGALY	S1 AND S2 PRESENT. NO ADDED SOUNDS	DTR PRESENT	B/L DOWN	PRESENT	NO DIABETIC RETINOPATHY	9.1	30	319	36	8.9	N - 66.5%, L- 24.4 %, M - 7.9 %, E- 0.8%, B- 0.4%	82	7.68	6
20	NVBS. NO ADDED SOUNDS	SOFT, NON-TENDER. NO ORGANOMEGALY	S1 AND S2 PRESENT. NO ADDED SOUNDS	DTR PRESENT	B/L DOWN	PRESENT	SEVERE NPDR	8.3	25.6	150	34	8.4	N - 73%, L- 17.2%, M - 3.9% %, E- 5.5%, B- 0.4%	144	9.1	9
21	NVBS. NO ADDED SOUNDS	SOFT, NON-TENDER. NO ORGANOMEGALY	S1 AND S2 PRESENT. NO ADDED SOUNDS	DTR PRESENT	B/L DOWN	PRESENT	NO DIABETIC RETINOPATHY	10.2	32.7	298		6.3	N - 69%, L- 17.4 %, M - 12.8 %, E- 0.4%, B- 0.4%	137	1.95	14.6
22	NVBS. NO ADDED SOUNDS	SOFT, NON-TENDER. NO ORGANOMEGALY	S1 AND S2 PRESENT. NO ADDED SOUNDS	DTR PRESENT	B/L DOWN	PRESENT	NO DIABETIC RETINOPATHY	9.6	29.3	319		4.9	N - 69%, L- 17.4 %, M - 12.8 %, E- 0.4%, B- 0.4%	268	2.15	12.6
23	NVBS. NO ADDED SOUNDS	SOFT, NON-TENDER. NO ORGANOMEGALY	S1 AND S2 PRESENT. NO ADDED SOUNDS	DTR PRESENT	B/L DOWN	PRESENT	NO DIABETIC RETINOPATHY	9.3	28.2	248	68	8.4	N - 77.1%, L- 19.9%, M - 11% %, E- 0.3%, B- 0.4%	222	7.88	32.4
24	NVBS. NO ADDED SOUNDS	SOFT, NON-TENDER. NO ORGANOMEGALY	S1 AND S2 PRESENT. NO ADDED SOUNDS	DTR PRESENT	B/L DOWN	PRESENT	NO DIABETIC RETINOPATHY	7.8	25.3	154		7.6	N - 77.1%, L- 19.9%, M - 11% %, E- 0.3%, B- 0.4%	121	7.01	2.9
25	NVBS. NO ADDED SOUNDS	SOFT, NON-TENDER. NO ORGANOMEGALY	S1 AND S2 PRESENT. NO ADDED SOUNDS	DTR PRESENT	B/L DOWN	PRESENT	MODERATE NPDR	11.2	34	248		9.4	N - 77.1%, L- 19.9%, M - 11% %, E- 0.3%, B- 0.4%	72	2.5	5
26	NVBS. NO ADDED SOUNDS	SOFT, NON-TENDER. NO ORGANOMEGALY	S1 AND S2 PRESENT. NO ADDED SOUNDS	DTR PRESENT	B/L DOWN	PRESENT	NO DIABETIC RETINOPATHY	9.6	31	274	120	7.4	N - 66.5%, L- 24.4 %, M - 7.9 %, E- 0.8%, B- 0.4%	64	4.39	10.2
27	NVBS. NO ADDED SOUNDS	SOFT, NON-TENDER. NO ORGANOMEGALY	S1 AND S2 PRESENT. NO ADDED SOUNDS	DTR PRESENT	B/L DOWN	PRESENT	NO DIABETIC RETINOPATHY	8.7	23.3	298		11.6	N - 73%, L- 17.2%, M - 3.9% %, E- 5.5%, B- 0.4%	154	3.17	10
28	NVBS. NO ADDED SOUNDS	SOFT, NON-TENDER. NO ORGANOMEGALY	S1 AND S2 PRESENT. NO ADDED SOUNDS	DTR PRESENT	B/L DOWN	PRESENT	NO DIABETIC RETINOPATHY	9.8	31	170		5.14	N - 77.1%, L- 19.9%, M - 11% %, E- 0.3%, B- 0.4%	85	1.93	7
29	NVBS. NO ADDED SOUNDS	SOFT, NON-TENDER. NO ORGANOMEGALY	S1 AND S2 PRESENT. NO ADDED SOUNDS	DTR PRESENT	B/L DOWN	PRESENT	NO DIABETIC RETINOPATHY	9	45	170	20	9.4	N - 80.1%, L- 19.9%, M - 11% %, E- 0.3%, B- 0.4%	200	4.5	8
30	NVBS. NO ADDED SOUNDS	SOFT, NON-TENDER. NO ORGANOMEGALY	S1 AND S2 PRESENT. NO ADDED SOUNDS	DTR PRESENT	B/L DOWN	PRESENT	NO DIABETIC RETINOPATHY	8.4	27.9	150		9.4	N - 80.1%, L- 19.9%, M - 11% %, E- 0.3%, B- 0.4%	116	5.49	10.5
31	NVBS. NO ADDED SOUNDS	SOFT, NON-TENDER. NO ORGANOMEGALY	S1 AND S2 PRESENT. NO ADDED SOUNDS	DTR PRESENT	B/L DOWN	PRESENT	SEVERE NPDR	8.1	23.5	335		10.75	N - 80.1%, L- 19.9%, M - 9% %, E- 0.3%, B- 0.6%	154	8.45	6
32	NVBS. NO ADDED SOUNDS	SOFT, NON-TENDER. NO ORGANOMEGALY	S1 AND S2 PRESENT. NO ADDED SOUNDS	DTR PRESENT	B/L DOWN	PRESENT	NO DIABETIC RETINOPATHY	10.5	34.5	287		11.6	N - 71.1%, L- 18.4 %, M - 9.3%, E- 1.0%, B- 0.4%	104	6.22	16.6
33	NVBS. NO ADDED SOUNDS	SOFT, NON-TENDER. NO ORGANOMEGALY	S1 AND S2 PRESENT. NO ADDED SOUNDS	DTR PRESENT	B/L DOWN	PRESENT	NO DIABETIC RETINOPATHY	10	31.3	181		6.2	N - 62.7%, L- 20.9%, M - 14% %, E- 2.3%, B- 0.4%	154	2.58	15.97
34	NVBS. NO ADDED SOUNDS	SOFT, NON-TENDER. NO ORGANOMEGALY	S1 AND S2 PRESENT. NO ADDED SOUNDS	DTR PRESENT	B/L DOWN	PRESENT	NO DIABETIC RETINOPATHY	8.1	27.5	130	82	7.4	N - 62.7%, L- 20.9%, M - 14% %, E- 2.3%, B- 0.4%	84	2.74	15
35	B/L DECREASED BREATH SOUNDS	SOFT, NON-TENDER. NO ORGANOMEGALY	S1 AND S2 PRESENT. NO ADDED SOUNDS	DTR PRESENT	B/L DOWN	PRESENT	NO DIABETIC RETINOPATHY	9.9	30.9	170		9.4	N - 80.1%, L- 19.9%, M - 11% %, E- 0.3%, B- 0.4%	84	6.25	6.23

S.No	URINE ROUTINE PROTEIN	URINE ROUTINE GLUCOSE	URINE ROUTINE PUS CELLS	URINE ROUTINE RBC	Sodium	Potassium	Chloride	Total cholesterol	TG	LDL	SGOT	SGPT	ALKALINE PHOSPHATASE	TOTAL BILIRUBIN	DIRECT BILIRUBIN	ALBUMIN	TOTAL PROTEIN	Calcium	Phosphorus
18	3+	2+	0	0	141	4.21	100				9	20	76	0.19	0.07	3.71	3.8	9.6	6
19	3+	2+	0	2	139	4.59	109	216	200	40	40	43	129	0.4	0.26	3.71	4.6	9.8	6
20	3+	3+	0	0	129	4.21	100	200			20	24	67	2	0.3	3.2	5	8.2	6
21	4+	0	3	10	136	3.3	100	225			14	11	96	0.55	0.1	3.1	4.2	9.1	4
22	4+	1+	0	0	138	4.3	109				21	32	45	0.2	0.1	3.2	4.6		
23	4+	3+	0	7	138	4.21	100				12	15	44	0.1	0.2	3.4	4.7	8	8.3
24	2+	2+	0	121.8	136	5.52	100	86	86	36	49	35	50	0.6	0.1	3	3.8	8.3	5.6
25	2+	2+	0	0	139	5.52	107				14	12	99	0.7	0.8	3	6	7.8	3.9
26	3+	2+	0	4	136	4.21	108				12	17	34	0.2	0.3	3.2	4	8.2	5.5
27	2+	2+	0	12.6	136	5.52	107				5	21	80	0.09	0.03	3.4	3.2	8	4
28	2+	3+	2	0	141	3.74	109	216	200	40	46	37	50	0.1	0.3	3	5	7.9	4.5
29	0	0	0	0	138	4.59	110				12	11	66	0.6	0.1	3.1	2.5	8.6	7
30	3+	1+	0	2.6	141	4.42	100				22	23	100	0.12	0.13	3.1	2.2	7.2	5.91
31	2+	2+	0	0	136	5.52	99	206			371	686	120	0.6	0.1	3.1	4	8.9	4.4
32	4+	2+	0	0	138	3.74	100				14	11	96	0.55	0.1	3.7	4.2	9.1	4
33	4+	1+	2	0	143	4.21	111				43	21	66	0.6	0.1	1.76	2.68	7.6	6.24
34	3+	1+	2	55.2	141	5.52	111				24	27	65	0.6	0.1	2	3		
35	3+	2+	0	1	141	5.52	107				12	16	45	0.2	0.3	2.89	6.2	7.2	7

S.No	Uric acid	Viral markers	HBA1c	USG abdomen	Others:	Histopathology report: Immuno-florescence (if needed)	Histopathology Impression:	NDKD	Final diagnosis	Outcome:	Treatment plan	Treatment started - Yes/No
18		NON-REACTIVE	8	ACUTE MEDICAL RENAL DISEASE GRADE	NOT DONE	NO GLOMERULAR DEPOSITS SEEN.	DKD	N	DIABETIC NEPHROPATHY	IMPROVED	STRICT GLYCEMIC CONTROL	YES
19	6.6	NON-REACTIVE	7.2	ACUTE MEDICAL RENAL DISEASE	NOT DONE	NO GLOMERULAR DEPOSITS SEEN.	mixed	N	DIABETIC NEPHROPATHY WITH FEATURES OF ACUTE PYELONEPHRITIS	IMPROVED	STRICT GLYCEMIC CONTROL AND ANTIBIOTICS	YES
20		NON-REACTIVE	NOT DONE	BILATERAL RENAL PARENCHYMAL CHANGES	NOT DONE	MESANGIAL POSITIVITY OF IGA (+2) EVIDENT	NDKD	Y	IGA NEPHROPATHY	IMPROVED	STEROIDS	YES
21	6	NON-REACTIVE	9	BILATERAL RENAL PARENCHYMAL CHANGES	NOT DONE	FULL HOUSE POSITIVITY	NDKD	N	LUPUS NEPHRITIS	IMPROVING	STEROIDS	YES
22		NON-REACTIVE	NOT DONE	NOT DONE	NOT DONE	DEPOSITS ARE POSITIVE FOR KAPPA (2+)	NDKD	Y	RENAL AMYLOIDOSIS	IMPROVED	-	YES
23	7.3	NON-REACTIVE	NOT DONE	NO SIGNIFICANT FINDINGS	NOT DONE	DIF CORE SHOWS 2 SCLEROSED GLOMERULI	NDKD	Y	TUBULO-INTERSTITIAL NEPHRITIS	IMPROVED	STEROIDS	YES
24		NON-REACTIVE	8.8	BILATERAL RENAL PARENCHYMAL CHANGES	ANACA PROFILE POSITIVE	DIF CORE SHOWS 5 SCLEROSED GLOMERULI	NDKD	Y	RAPIDLY PROGRESSIVE GLOMERULO NEPHRITIS	IMPROVED	PULSE STEROID THERAPY	YES
25	5.6	NON-REACTIVE	8.8	NOT DONE	NOT DONE	NOT SIGNIFICANT	mixed	N	DIABETIC NEPHROPATHY WITH ACUTE TUBULO-INTERSTITIAL NEPHRITIS	IMPROVED	STRICT GLYCEMIC CONTROL	YES
26	5.9	NON-REACTIVE	7.3	NO SIGNIFICANT FINDINGS	NOT DONE	NO GLOMERULAR DEPOSITS SEEN.	NDKD	Y	TUBULO-INTERSTITIAL NEPHRITIS	IMPROVED	STEROIDS	YES
27	6.6	NON-REACTIVE	9.7	NOT DONE	NOT DONE	NO GLOMERULAR DEPOSITS SEEN.	NDKD	Y	GLOMERULO NEPHRITIS	IMPROVED	STEROIDS	YES
28	5.7	NON-REACTIVE	9	BILATERAL MEDICAL RENAL DISEASE	NOT DONE	NO GLOMERULAR DEPOSITS SEEN.	DIABETIC KIDNEY DISEASE	N	DIABETIC NEPHROPATHY	IMPROVED	STRICT GLYCEMIC CONTROL	YES
29	5.6	NON-REACTIVE	8	NOT DONE	NOT DONE	18 SCLEROSED GLOMERULI	DIABETIC KIDNEY DISEASE	N	DIABETIC NEPHROPATHY	IMPROVED	STRICT GLYCEMIC CONTROL AND BP CONTROL	YES
30	7.3	NON-REACTIVE	NOT DONE	ACUTE MEDICAL RENAL DISEASE	ANA POSITIVE ANCA PROFILE NEGATIVE	NO GLOMERULAR DEPOSITS SEEN.	DIABETIC KIDNEY DISEASE	N	DIABETIC NEPHROPATHY	IMPROVED	STRICT GLYCEMIC CONTROL	YES
31	5.3	NON-REACTIVE	11.4	ACUTE MEDICAL RENAL DISEASE	ANA NEGATIVE ANCA PROFILE NEGATIVE	NO GLOMERULAR DEPOSITS SEEN.	NDKD	Y	ATN	IMPROVED	CONSERVATIVE MANAGEMENT	YES
32	4.4	NON-REACTIVE	NOT DONE	NOT DONE	NOT DONE	NO GLOMERULAR DEPOSITS SEEN.	DIABETIC KIDNEY DISEASE	N	DIABETIC NEPHROPATHY	IMPROVED	STRICT GLYCEMIC CONTROL	YES
33	4.4	NON-REACTIVE	9	ACUTE MEDICAL RENAL DISEASE	NOT DONE	NO GLOMERULAR DEPOSITS SEEN.	NDKD	Y	ACUTE TUBULAR INJURY	IMPROVED	CONSERVATIVE MANAGEMENT	YES
34		NON-REACTIVE	NOT DONE	NOT DONE	NOT DONE	NO GLOMERULAR DEPOSITS SEEN.	NDKD	Y	DIFFUSE PROLIFERATIVE GLOMERULONEPHRITIS	IMPROVED	STERORIDS AND CYCLOPHOSPHAMIDE	YES
35	6.4	NON-REACTIVE	7	BILATERAL RENALL PARENCHYMAL CHANGES	ANA NEGATIVE ANCA NEGATIVE	NO GLOMERULAR DEPOSITS SEEN.	MIXED NDKD AND DKD	N	DIABETIC NEPHROPATHY AND ACUTE TUBULO-INTERSTITIAL NEPHRITIS	IMPROVED	STRICT GLYCEMIC CONTROL	YES

S.No	Name	MRD number	Age	AGE GROUP	Sex	Occupation	Date of admission	History of present illness_Dyspnoea (Yes/No)	Chest pain (Yes/No)	Decreased urine output (Yes/No)	Facial puffiness (Yes/No)	Oedema feet (Yes/No)	Haematuria (Yes/No)	Any other symptoms:	Drug intake (NSAIDS, Antibiotics, Indigenous medications etc) (Yes/No)
36	DS	1423825	51	3	MALE	FARMING	30-11-2022	NO	NO	NO	YES	YES	NO	COUGH	NO
37	JS	1403484	55	3	MALE	SHOPKEEPER	18-11-2022	YES	NO	NO	YES	YES	NO	FEVER	NO
38	GS	1501161	65	4	MALE	RETIRED	18-04-2023	NO	NO	YES	YES	NO	NO	NO	NO
39	MS	1396785	34	1	FEMALE	HOUSEWIFE	12-05-2023	NO	NO	NO	NO	YES	NO	FROTHURIA	NO
40	KB	1457272	50	2	FEMALE	HOUSEWIFE	18-04-2023	NO	NO	YES	NO	NO	NO	LOWER LIMB PAIN	NO
41	VKS	1674629	75	5	MALE	RETIRED	22-02-2024	NO	NO	NO	NO	YES	NO	POOR URINE STREAM	NO
42	SS	1658688	70	4	MALE	RETIRED	25-01-2024	NO	NO	NO	NO	YES	NO	FEVER	NO
43	SG	1638304	46	2	FEMALE	HOUSEWIFE	13-12-2023	NO	NO	NO	NO	NO	NO	WEIGHT LOSS	NO

S.No	Diabetes Mellitus Number of years:	Diabetes Mellitus On regular treatment (Yes/No)	Hypertension Number of years:	Hypertension On regular treatment (Yes/No)	Any other relevant past history:	Smoking (Yes/No)	Alcohol (Yes/No)	Any other drug abuse (Yes/No)	BMI (kg/m2)	Pulse rate (x/min)	SBP	DBP	Respiratory rate:	Cyanosis:	Clubbing:	Icterus:	Pallor:	JVP:
36	12	YES	15 YEARS	YES	OLD CVA	NO	NO	NO	24	90	130	80	18	NO	NO	NO	NO	NO
37	15	YES	2 YEARS	YES	NO	NO	NO	NO	28	80	130	80	18	NO	NO	NO	NO	NO
38	6	YES	YES	NO	NO	NO	NO	NO	22	85	120	70	16	NO	NO	NO	NO	NO
39	7	YES	7 YEARS	YES	NO	NO	NO	NO	24	90	110	70	18	NO	NO	NO	NO	NO
40	9	YES	5 YEARS	YES	NO	NO	NO	NO	26	80	140	90	18	NO	NO	NO	NO	NO
41	15	YES	15 YEARS	YES	NO	NO	NO	NO	22	85	120	70	18	NO	NO	NO	NO	NO
42	20	YES	NO	NO	NO	NO	NO	NO	25	80	110	70	16	NO	NO	NO	NO	NO
43	7	YES	7 YEARS	YES	NO	NO	NO	NO	24	70	130	80	18	NO	NO	NO	NO	NO

S.No	Respiratory exam:	Abdominal exam	Cardiovascular exam	Central nervous system exam Reflexe	Plantar	Vibration sense	Fundus	HB	PCV	Plateletc ount	ESR	TLC	DLC	Blood urea	S. creatinine	UPCR
36	NVBS. NO ADDED SOUNDS	SOFT, NON-TENDER. NO ORGANOMEGALY	S1 AND S2 PRESENT. NO ADDED SOUNDS	DTR PRESENT	B/L DOWN	PRESENT	MODERATE NPDR	13	40	170	73	11.6	N - 73%, L- 17.2%, M - 3.9% %, E- 5.5%, B- 0.4%	128	2.38	7.4
37	NVBS. NO ADDED SOUNDS	SOFT, NON-TENDER. NO ORGANOMEGALY	S1 AND S2 PRESENT. NO ADDED SOUNDS	DTR PRESENT	B/L DOWN	PRESENT	NO DIABETIC RETINOPATHY	10.2	30	248		7.6	N - 62.7%, L- 20.9%, M - 14% %, E- 2.3%, B- 0.4%	53	1.3	12.5
38	NVBS. NO ADDED SOUNDS	SOFT, NON-TENDER. NO ORGANOMEGALY	S1 AND S2 PRESENT. NO ADDED SOUNDS	DTR PRESENT	B/L DOWN	PRESENT	NO DIABETIC RETINOPATHY	10.5	32	248		5.14	N - 62.7%, L- 20.9%, M - 14% %, E- 2.3%, B- 0.4%	90	4.6	14
39	NVBS. NO ADDED SOUNDS	SOFT, NON-TENDER. NO ORGANOMEGALY	S1 AND S2 PRESENT. NO ADDED SOUNDS	DTR PRESENT	B/L DOWN	PRESENT	NO DIABETIC RETINOPATHY	8.9	29.5	148	44	8.4	N - 62.7%, L- 20.9%, M - 14% %, E- 2.3%, B- 0.4%	90	1.4	8.8
40	NVBS. NO ADDED SOUNDS	SOFT, NON-TENDER. NO ORGANOMEGALY	S1 AND S2 PRESENT. NO ADDED SOUNDS	DTR PRESENT	B/L DOWN	PRESENT	MODERATE NPDR	7.5	23	335		9.4	N - 80.1%, L- 19.9%, M - 9% %, E- 0.3%, B- 0.6%	100	4.37	9
41	NVBS. NO ADDED SOUNDS	SOFT, NON-TENDER. NO ORGANOMEGALY	S1 AND S2 PRESENT. NO ADDED SOUNDS	DTR PRESENT	B/L DOWN	PRESENT	NO DIABETIC RETINOPATHY	10.7	30	248		9.4	N - 80.1%, L- 19.9%, M - 9% %, E- 0.3%, B- 0.6%	47	2.12	9
42	NVBS. NO ADDED SOUNDS	SOFT, NON-TENDER. NO ORGANOMEGALY	S1 AND S2 PRESENT. NO ADDED SOUNDS	DTR PRESENT	B/L DOWN	PRESENT	NO DIABETIC RETINOPATHY	9.4	29	310		7.6	N - 71.1%, L- 18.4 %, M - 9.3%, E- 1.0%, B- 0.4%	137	6.57	10
43	NVBS. NO ADDED SOUNDS	SOFT, NON-TENDER. NO ORGANOMEGALY	S1 AND S2 PRESENT. NO ADDED SOUNDS	DTR PRESENT	B/L DOWN	PRESENT	SEVERE NDPR	9.8	29	226		8.4	N - 73%, L- 17.2%, M - 3.9% %, E- 5.5%, B- 0.4%	80	6.66	8.9

S.No	URINE ROUTINE PROTEIN	URINE ROUTINE GLUCOSE	URINE ROUTINE PUS CELLS	URINE ROUTINE RBC	Sodium	Potassium	Chloride	Total cholesterol	TG	LDL	SGOT	SGPT	ALKALINE PHOSPHATASE	TOTAL BILIRUBIN	DIRECT BILIRUBIN	ALBUMIN	TOTAL PROTEIN	Calcium	Phosphorus
36	3+	4+	0	4	129	3.74	81				50	35	50	0.6	0.1	3	3.8		
37	3+	3+	0	0	141	4.98	108	128	96	76	24	23	54	0.1	0.3	3	4.8		
38	3+	2+	0	0	138	3.27	109				12	13	43	0.2	0.3	2.89	6.2	9.4	3.9
39	3+	2+	0	0	136	4.3	108	140			22	23	66	0.15	0.11	3.1	2.68		
40	2+	3+	0	0	129	4.59	99				11	12	54	0.6	0.1	3.1	2.68	9.6	4.5
41	3+	1+	0	4	138	4.3	100				14	12	96	0.55	0.1	3.1	4.2	8.3	4
42	3+	3+	0	4	143	4.42	107				12	15	45	0.2	0.3	3.4	4.6	9.1	5.12
43	3+	3+	0	3	136	3.27	115				40	45	44	0.16	0.15	3.1	2.68		

S.No	Uric acid	Viral markers	HBA1c	USG abdomen	Others:	Histopathology report: Immuno-florescence (if needed)	Histopathology Impression:	NDKD	Final diagnosis	Outcome:	Treatment plan	Treatment started - Yes/No
36		NON-REACTIVE	NOT DONE	BILATERAL RENAL PARENCHYMAL CHANGES	ANA PROFILE NEGATIVE	NO GLOMERULAR DEPOSITS SEEN.	DIABETIC KIDNEY DISEASE	N	DIABETIC NEPHROPATHY	IMPROVED	STRICT GLYCEMIC CONTROL	YES
37		NON-REACTIVE	NOT DONE	ACUTE MEDICAL RENAL DISEASE	NOT DONE	NO GLOMERULAR DEPOSITS SEEN.	mixed	Y	FOCAL PROLIFERATIVE GLOMERULONEPHRITIS. DIABETIC NEPHROPATHY AND ACURE INTERSTITIAL NEPHRITIS	IMPROVED	STEROIDS AND STRICT GLYCEMIC CONTROL	YES
38	5.6	NON-REACTIVE	NOT DONE	ACUTE MEDICAL RENAL DISEASE	NOT DONE	6 VIABLE AND 4 SCLEROSED GLOMERULI	NDKD	Y	FOCAL SEGMENTAL GLOMERULOSCLEROSIS.	IMPROVED	STRICT GLYCEMIC CONTROL AND HTN CONTROL	YES
39		NON-REACTIVE	8	NOT DONE	ANA PROFILE NEGATIVE	3-4 SCLEROSED GLOMERULI	DIABETIC KIDNEY DISEASE	N	DIABETIC NEPHROPATHY	IMPROVED	STRICT GLYCEMIC CONTROL	YES
40	5.7	NON-REACTIVE	5.9	NOT DONE	NOT DONE	NO GLOMERULAR DEPOSITS SEEN.	NDKD	Y	FOCAL PROLIFERATIVE GLOMERULO-NEPHRITIS	IMPROVED	STEROIDS AND STRICT GLYCEMIC CONTROL	YES
41		NON-REACTIVE	NOT DONE	NO SIGNIFICANT FINDINGS	NOT DONE	NON-SPECIFIC TRAPPING OF IGM SEEN IN GLOMERULI	DKD	N	DIABETIC NEPHROPATHY	IMPROVED	STRICT GLYCEMIC CONTROL	YES
42		NON-REACTIVE	NOT DONE	BILATERAL RENAL PARENCHYMAL CHANGES	ANA POSITIVE ANCA PROFILE NEGATIVE	STRONG 2+ COARSE GRANULAR STAINING FOR C3C AND 1+ STAINING FOR IGA	NDKD	Y	IMMUNE COMPLEX MEDIATED GLOMERULONEPHRITIS -IGA DOMINATE INFECTION RELATED GLOMERULONEPHRITIS	IMPROVED	STEROIDS	YES
43		NON-REACTIVE	NOT DONE	BILATERAL RENAL PARENCHYMAL CHANGES	NOT DONE	NO GLOMERULAR DEPOSITS SEEN.	NDKD	Y	ACUTE PYELONEPHRITIS	IMPROVED	ANTIBIOTICS	YES

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No. BFUHS/2K23p-TH/ 13224 **Date** 29/12/23

The Principal
Dayanand Medical College & Hospital
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Subject:: Approval of Plan of Thesis.

This is to inform you that the Faculty of Medical Sciences in its meeting held on 25.09.2023 has approved the plan of thesis of **Rohit Ghai**, a student of your college for **MD General Medicine** along with the names of Supervisor/Co-Supervisor as under:-

Topic Plan of Thesis
Non-diabetic kidney diseases in patients with diabetes mellitus (clinicpathological correlation) .

Supervisor	**Co-Supervisor:-**	**Co-Supervisor:-**
Dr. Harmeet Pal Singh Dhooria	Dr. Suman Sethi	Dr. Pavneet Kaur Selhi
Assoc. Prof.,	Assoc. Professor	Professor
Dept. of Medicine	Dept. of Nephrology	Dept. of Pathology

The student may submit the thesis in triplicate alongwith the plan in triplicate also, to the Controller of Examinations, BFUHS, Faridkot, at least six months before the commencement of theory examination, if he/she is otherwise eligible to take the examination. He /She may be directed to attach a photocopy of this letter with the forwarding letter while submitting thesis in the University along with the letter of approval of University for change of supervisor/Co-Supervisor, change of topic, if any.

Controller of Examinations

Note :

1. *If any discrepancy found regarding topic of thesis or Supervisor/Co-Supervisor, inform undersigned within one month after issue of this letter.*

NON-DIABETIC KIDNEY DISEASES IN PATIENTS WITH DIABETES MELLITUS (CLINICOPATHOLOGICAL CORRELATION)

THESIS PROPOSAL

FOR APPROVAL OF SUBJECT OF THESIS TO BE SUBMITTED IN

PARTIAL FULFILMENT OF THE REQUIREMENTS

FOR THE DEGREE OF

MD

IN

GENERAL MEDICINE

OF THE

BABA FARID UNIVERSITY OF HEALTH SCIENCES

FARIDKOT

AUGUST, 2022

DR. ROHIT GHAI

DEPARTMENT OF GENERAL MEDICINE
DAYANAND MEDICAL COLLEGE AND HOSPITAL
LUDHIANA

ABSTRACT OF PLAN OF THESIS

Title	NON- DIABETIC KIDNEY DISEASES IN PATIENTS WITH DIABETES MELLITUS (CLINICOPATHOLOGICAL CORRELATION)
For the degree of	MD General Medicine
Name of the candidate	Dr. Rohit Ghai
Supervisor	Dr. Harmeet Pal Singh Dhooria, Associate Professor, Department of Medicine, DMCH, Ludhiana
Co-Supervisor	Dr. Suman Sethi, Associate Professor, Department of Nephrology, DMCH, Ludhiana Dr. Pavneet Kaur Selhi, Professor, Department of Pathology, DMCH, Ludhiana
Institution	Dayanand Medical College and Hospital,Ludhiana
University	Baba Farid University of Health Sciences, Faridkot

Introduction: Proteinuria or renal failure in diabetic patients is usually interpreted as a clinical manifestation of DN. However, not all diabetic subjects with proteinuria or renal failure have DN. NDKD, though rarer than DN, have been seen to cause proteinuria or renal failure in diabetics. Most patients with diabetic nephropathy are not formally evaluated with a renal biopsy. Kidney biopsy can differentiate DN from non-diabetic renal diseases (NDKD), but it is invasive. Some NDKDs diagnosed on kidney biopsy such as minimal change disease, immunoglobulin A (IgA) nephropathy and membranous nephropathy are often treatable and even curable.

Keywords: Diabetes mellitus, non-diabetic kidney disease

Aim of Study: To study the prevalence and clinical predictors of NDKD in Diabetes Mellitus patients.

Materials & methods: This will be a cross sectional observational study that will be conducted in Dayanand Medical College and Hospital, Ludhiana. Patients suspected of NDKD in patients of diabetes will undergo kidney biopsy after informed consent. *NDKD will be suspected with any of the below criteria, singly or in combination*:

1. Nephrotic range proteinuria or kidney failure in the absence of diabetic retinopathy.
2. Nephrotic range proteinuria or kidney failure with diabetes duration less than 5 years.
3. Nephrotic range proteinuria with normal kidney function.
4. Unexplained microscopic hematuria.
5. Acute kidney injury.
6. Rapidly worsening kidney function in patients with previously stable kidney function.

(*Reference: Brenner and Rector's The Kidney*)

(Signature) Candidate | (Signature) Co-supervisor | (Signature) Co-supervisor | (Signature) Supervisor | (Signature) Principal

CERTIFICATE OF FACILITIES AVAILABLE

This is to certify that the facilities for work on the subject of thesis titled **"NON- DIABETIC KIDNEY DISEASES IN PATIENTS WITH DIABETES MELLITUS (CLINICO PATHOLOGICAL CORRELATION)"** exist in the Department of General Medicine at Dayanand Medical College and Hospital, Ludhiana and will be provided to the candidate. We will see that the data being included in the thesis are genuine and is collected by the candidate himself under our supervision and guidance.

Supervisor

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Co-Supervisor's

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Place : _____

Date : _____

APPROVAL PROFORMA
BY
RESEARCH & ETHICAL COMMITTEE
DAYANAND MEDICAL COLLEGE & HOSPITAL, LUDHIANA

Name of candidate	Dr. Rohit Ghai
Department	General Medicine
Topic of Thesis	NON- DIABETIC KIDNEY DISEASES IN PATIENTS WITH DIABETES MELLITUS (CLINICOPATHOLOGICAL CORRELATION)
Likely date of appearing in PG examination	May / June, 2024
Date of enrollment	Feb 7,2022
Name of Head of Department	Dr. Dinesh Gupta
1) Supervisor **2) Co-Supervisor (s)**	Dr. Harmeet Pal Singh Dhooria Dr. Suman Sethi Dr. Pavneet Kaur Selhi
Signature of Members of Research Committee with Stamp	**Signature of Members of Ethics Committee with Stamp**
Dr. Harpreet Kaur (Professor & Head, Pathology)	Mr. R K Sharma (Legal Expert)
Dr. Jaspal Singh (Professor & Head, Surgery)	Dr. Ramit Mahajan (Associate Professor, Gastroenterology)
Dr. Dinesh Gupta (Professor & Head, Medicine)	Dr. Anu Sharma (Professor, Anatomy)
Dr. Sandeep Kaushal (Dean Academics & Professor & Head, Pharmacology)	Dr. Sandeep Kaushal (Dean Academics & Professor & Head, Pharmacology)
Dr. Gurpreet S Wander (Vice Principal)	Dr. Gurpreet S Wander (Vice Principal & Professor & Head, Cardiology)
	Dr. Gagandeep Singh (Secretary, Ethics Committee Professor & Head, Neurology)

Approved: Yes / ~~No~~

Dr. Sandeep Puri
Chairperson
Research Committee

Chairperson
Research Committee

Approved: Yes / ~~No~~

Prof. Arvind Malhotra
Chairperson
Ethics Committee

Chairperson
Ethics Committee

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PROFORMA FOR REGISTRATION OF SUBJECT FOR DISSERTATION

1	NAME OF THE CANDIDATE AND ADDRESS	DR. ROHIT GHAI 1305 ARYA MOHALLA, MATA RANI CHOWK, CLOCK TOWER, LUDHIANA
2	NAME OF THE INSTITUTE	DAYANAND MEDICAL COLLEGE AND HOSPITAL,LUDHIANA
3	COURSE OF STUDY AND SUBJECT	MD, GENERAL MEDICINE
4	DATE OF ADMISSION OF COURSE	MARCH 7,2022
5	TITLE OF THE TOPIC	NON-DIABETIC KIDNEY DISEASE IN PATIENTS WITH DIABETES MELLITUS (CLINICO PATHOLOGICAL CORRELATION)

6. BRIEF RESUME OF THE INTENDED WORK

6.1 NEED FOR THE STUDY

DKD is the commonest cause of chronic kidney disease and end stage kidney disease worldwide. Consequently, it has become an important productive implication to the healthcare system. Around 20-40% patients with DM develop DKD. However, there are substantial number of diabetic patients who are diagnosed with non-diabetic kidney disease (NDKD), reaching as a high as 64-85% in some studies of biopsied patients. The diagnosis of DKD in a bulk of patients remains clinical and invasive tests like renal biopsy are usually reserved for atypical cases. While such, an approach is well validated in Type 1 Diabetes Mellitus, a rethinking of this approach may be required in Type 2 Diabetes Mellitus, given the often-high prevalence of NDKD.

The precise diagnosis of this non-diabetic renal disease has prognostic and therapeutic implications and it can be difficult to differentiate between NDKD and Diabetic Nephropathy clinically. It is important to differentiate between Diabetic nephropathy and NDKD because their therapy and prognosis are different.

Diabetic Nephropathy is hard to reverse but some NDKD are often treatable and even remittable. NDKD warrants special attention because when pick up early interventions can often reduce the associated morbidity, mortality and quality of life. Kidney biopsy is a gold standard in diagnosis of non-diabetic renal disease in diabetic patients; it is a guidepost for treatment and a prognostic indicator too.

Kidney biopsy can find out that a patient has a) Diabetic Nephropathy b) Non-diabetic renal disease alone c) NDKD superimposed on Diabetic Nephropathy.

NDKD are an important cause of renal involvement in diabetics but few research works focusing on this entity have been done. So aim of carrying out this study is to analyze the prevalence and clinical predictors of NDKD in type 2 diabetes mellitus patients.

6.2 REVIEW OF LITERATURE

Diabetic kidney disease (DKD) is one of the most significant micro vascular complications of diabetes and is the leading cause of end-stage kidney disease (ESKD) requiring renal replacement therapy in adults. However, in diabetic patients kidney diseases other than DKD can develop. The prevalence of non-diabetic kidney disease (NDKD) is reported at different rates in the biopsied diabetic cohorts. NDKD can occur either alone or superimposed on DKD. It is important to diagnose NDKD in diabetics because the treatment and prognosis of these diseases are quite different. The diagnosis of DKD is usually based on clinical features, clinicians' and/or single-center experiences. Among diabetic patients, the indications of kidney biopsy are debatable, and it is not performed routinely. Therefore, the correct diagnosis of such patients would be crucial for disease specific therapy.

Eswarappa M et al (2022): A retrospective observational study was conducted on the patients who were admitted between January 2012 and June 2017 in a tertiary hospital of South India to assess the prevalence of NDKD in type 2 Diabetic Mellitus patients using biopsy findings. Out of 236 renal biopsies 78 had NDKD with diabetic nephropathy and 44 had isolated NDKD. Acute interstitial nephritis was most common cause of NDKD. Long duration of DM beyond 5 years and Hypertension beyond 2 years predict DKD.[1]

Arora P et al (2020): Hospital based prospective study was done on 44 patients with type 2 diabetes to analyze the incidence and spectrum of NDKD in patients with diabetes with or without diabetic nephropathy, effect of early specific interventions on outcome and renal-retinal relationship in type 1 and type 2 diabetes mellitus with nephropathy by doing renal biopsies. Out of 44 patients 61.4% had isolated NDKD, 13.6 % had NDKD superimposed on diabetic nephropathy, 25% had isolated diabetic nephropathy. Most common NDKD was minimal change disease. In NDKD group, 78.8% patients recovered functionally and clinically, 15% were dialysis dependent when discharged and 2 patients died during treatment. Accurate diagnosis by renal biopsy showed clinical improvement in significant number of patients.[2]

Souza DA et al: (2020): A retrospective study including kidney biopsies performed on 79 diabeticspatients between 1987 and 2013. NDKD were present in 41.77% of patients. Membranous GN (30.3%) and IGA nephropathy were the most common NDKD. Probability of presence of NDKD with or without concomitant diabetic nephropathy is greater for patients who had biopsies with a time since diagnosis of 5 years or less.[3]

Sharma M et al: (2020): Study is on patients of 140 type 2diabetics who were aged >18 years toevaluate the renal biopsies for suspicion of NDKD and to correlate clinico-pathological findings. Recent onset nephrotic syndrome was the most common indication for biopsy. NDKD was seen in 34% of patients, Diabetic nephropathy +/- NDKD in 24%, isolated diabetic nephropathy in 42%. Focal segmental glomerulosclerosis and IgA nephropathy were most common causes of isolated NDKD. Shorter duration of diabetes, absence of diabetic retinopathy and lower glycated hb were predictors of NDKD.[4]

Grujicic M et al: (2019): Study was done to investigate theincidence and type of NDKD diagnosed by kidney biopsy donebetween April 2007 to October 2018 inpatients will type 2 diabetes mellitusand correlation of clinical and laboratory findings with histopathological diagnosis. Biopsy indications were nephrotic syndrome, asymptomatic urinary abnormalities in 3 patients and rapid chronic renal failure progression. Out of 17 patients, 6 (35.3%) had NDKD, 3(17.6%) had NDKD superimposed with diabetic nephropathy and 8 (47.1%) had diabetic nephropathy. NDKD was diagnosed using kidney biopsy in patients with type 2 diabetes.[5]

Erdogmus S et al: (2017): A retrospective study by reviewing the kidney biopsies done in last 10 years to investigate the utility of kidney biopsy in patients with type 2 diabetes, predictability of diagnosing DKD versus NDKD from clinical and laboratory data and to evaluate the prevalence and etiology of NDKD in patients with type 2 diabetes mellitus. Out of 48 patients 24 (50%) patients had NDKD alone, 20(41.7%) had DKD alone and 4 (8.3%) had co-existing DKD and NDKD. Most common NDKD were membranous nephropathy (29.2%),

tubulointerstitial nephritis (20.8%) and IgA nephropathy (12.5%). Absence of diabetic retinopathy strongly predicted NDKD.[6]

Kanodia KV et al: (2017): A single center retrospective study byanalyzing renal biopsies of type 2 diabetic patients, performed between Jan 2008 and Sep 2016 to find clinical, laboratory and histological features of NDKD and to assess the prevalence of NDKD in India. Out of 152 diabetic patients, 35% (23.03%) patients had isolated NDKD, 35% (23.03%) had NDKD superimposed on underlying diabetic nephropathy and 82% (53.95%) had isolated diabetic nephropathy. Most common clinical presentation was nephrotic syndrome. Most common histological type of NDKD was acute tubulointerstitial nephritis. Shorter duration of diabetes, hematuria, absence of retinopathy, low serum complements levels and nephrotic range proteinuria are predictors of NDKD.[7]

Solemanian T et al (2015): A prospective cohort study was done on 46 patients with type 2 diabetes

mellitus and atypical features of diabetic nephropathy with unexpected proteinuria, hematuria and or renal impairment to identify underlying renal pathology. Out of 46, 16 (34.8%) patients had diabetic nephropathy, 20 (43.5%) had NDKD, and 10 (21.7%) had NDKD superimposed on diabetic nephropathy. Membranous nephropathy (34%) was the most common NDKD. Patients with NDKD had lower frequency of diabetic retinopathy (5%), shorter duration of diabetes, high range of proteinuria and better kidney survival. Kidney biopsy is strongly recommended for patients with type 2 diabetes and atypical presentation for diabetic nephropathy, particularly in absence of diabetic retinopathy.[8]

Wilfred DC et al: (2013): Study is done on 93 patients of type 2 diabetes mellitus and atypical clinicalrenal disease to know the frequency and spectrum of NDKD and common clinical markers that are associated with NDKD in local population by doingrenal biopsy. 68.8% patients had NDKD with or without concurrentdiabetic nephropathy. Shorter durationof diabetes, absence of retinopathy,

positive urine microscopic hematuria and active urinary sediment are

markers associated with type 2diabetes with clinical renal disease. Chronic interstitial nephritis was the most common NDKD.[9]

Yaqub S et al (2012): Retrospective study was done inpatients with type 2 diabetes mellituswho underwent kidney biopsy from Jan 2003 to Dec 2007 to evaluate the etiology of NDKD in patients with type 2 diabetes, Isolated NDKD was present in 34 patients (52%), 11 patients (17%) had NDKD with underlying diabetic nephropathy and 23 patients (31%) had isolated diabetic nephropathy. Most common NDKD were acute interstitial nephritis. Renal biopsy in diabetic and no extra renal end organ damage helps to diagnose and treat NDKD.[10]

6.3 AIMS AND OBJECTIVES OF THE STUDY

- To study the prevalence and clinical predictors of NDKD in diabetes mellitus patients.

7. MATERIALS AND METHODS

This will be a cross sectional observational study that will conducted in Dayanand Medical College and Hospital, Ludhiana. Patients suspected of NDKD in patients of diabetes undergo kidney biopsy after informed consent. NDKD will be suspected in patients of diabetes mellitus when they present with **any of the below mentioned criteria singly or in combination:**

1.Hematuria (RBC >5/hpf, RBC casts)

2.Sudden increase in serum creatinine by >2 mg/dL

3 Sudden onset nephrotic syndrome

4 Renal failure in absence of diabetic retinopathy (DR)

5 Massive proteinuria (nephrotic range) with normal renal functions

6 Significant renal insufficiency (serum creatinine >2 mg/dL) with normal or insignificant proteinuria. (<500 mg/dL)

These patients will be subjected to kidney biopsy to confirm the presence of NDKD.

7.1 SOURCE OF DATA: The study will be conducted in the department of Medicine, department of Nephrology and allied department of medicine at DMC and Hospital, Ludhiana.

7.2 METHOD OF COLLECTION OF DATA: Patients suspected of NDKD inpatients of diabetes undergokidney biopsy after informedconsent. NDKD will be suspected in patients of diabetes mellituswhen they **present with any of the below mentioned criteria singly or in combination:**

1. Nephrotic range proteinuria or kidney failure in the absence of diabetic retinopathy.

2. Nephrotic range proteinuria or kidney failure with diabetes duration less than 5 years.

3. Nephrotic range proteinuria with normal kidney function.

4. Unexplained microscopic hematuria.

5. Acute kidney injury.

6. Rapidly worsening kidney function in patients with previously stable kidney function.

These patients will be subjected to kidney biopsy to confirm the presence of NDKD.

Inclusion criteria: Patients suspected of NDKD in patients of type 2 diabetes mellitus that undergo kidney biopsy.

Exclusion criteria:

Patients will be excluded if they meet any of the following criteria:

1 Ongoing urinary tract infection

2 Calculus renal disease

3 Obstructive uropathy

4 Renal tumours

5 Bleeding diathesis

6 Bilateral contracted kidneys (kidney size of no less than 8cm)

7 Single contracted kidney (contraindication for biopsy)

8 Patients not consenting for kidney biopsy

9 Patients who don't give consent for study

DURATION OF STUDY

1.5 YEARS

OUTCOME

To analyze kidney biopsies and treat potentially reversible causes of NDKD.

STATISTICAL ANALYSIS

Data will be described in terms of range; mean ±standard deviation (± SD), frequencies (number of cases) and relative frequencies (percentages) as appropriate. All statistical calculations will be done using (Statistical Package for the Social Science) SPSS 21version (SPSS Inc., Chicago, IL, USA) statistical program for Microsoft Windows.

7.3 DOES THE STUDY REQUIRE ANY INVESTIGATIONS OR INTERVENTIONS TO BE CONDUCTED ON PATIENTS OR OTHER HUMANS OR ANIMALS? IF SO, PLEASE DESCRIBE BRIEFLY.

Yes, kidney biopsy.

Consent will be obtained from the patient prior to kidney biopsy. Indications and contraindications will be established. Under USG guidance, the left kidney will be identified. Under total aseptic conditions, local infiltration with 2% xylocaine will be done for anesthesia. Using a 18 french renal biopsy gun, two core tissues will be taken form the lower pole of the left kidney. Post biopsy precautions will be explained to the patient.

7.4 HAS ETHICAL CLEARANCE BEEN OBTAINED FROM YOUR INSTITUTION IN CASE OF 7.3 ?

Attached herewith.

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PROFORMA

S.No:

Name:

MRD number:

Age/sex:

Fathers name:

Address:

Occupation:

Date of admission:

History of present illness

Dyspnoea (Yes/No)

Chest pain (Yes/No)

Decreased urine output (Yes/No)

Facial puffiness (Yes/No)

Oedema feet (Yes/No)

Haematuria (Yes/No)

Any other symptoms:

Drug intake (NSAIDS, Antibiotics, Indigenous medications etc) (Yes/No)

Past History

Diabetes Mellitus

Number of years:

On regular treatment (Yes/No)

Hypertension

Number of years:

On regular treatment (Yes/No)

Any other relevant past history:

Personal history

Smoking (Yes/No)

Alcohol (Yes/No)

Any other drug abuse (Yes/No)

Examination

BMI:

Pulse rate:

Blood pressure:

Respiratory rate:

Cyanosis:

Clubbing:

Icterus:

Pallor:

JVP:

Systemic examination

Respiratory exam:

Abdominal exam:

Cardiovascular exam:

Central nervous system exam:

- Reflexes
- Plantar
- Vibration sense
- Fundus

Investigations

HB

PCV

Platelet count

ESR

TLC

DLC

Blood urea

S. creatinine

UPCR

Urine routine

S. electrolytes

- Sodium
- Potassium
- Chloride

PT/INR

Total cholesterol

TG

LDL

LFT

Calcium

Phosphorus

Uric acid

Additional investigations (if done):

1. Viral markers
2. HBA1c
3. USG abdomen
4. Others:

Histopathology report:

a. Immuno-florescence (if needed)

b. Histopathology

Impression:

Final diagnosis:

Outcome:

Treatment plan

Treatment started - Yes/No

INFORMED PATIENT CONSENT

Date: _____

I…….D/o, W/o, S/o……..…… am willing to participate in the study **"NON- DIABETIC KIDNEY DISEASES IN PATIENTS WITH DIABETES MELLITUS (CLINICO PATHOLOGICAL CORRELATION)"** The procedure with its complications has been explained to me in local vernacular language. I have fully understood that no special tests and extra expenditure in any form, merely for the purpose of thesis is being carried out. I am free to opt out the study any time without assigning any reason.

……. ……

Signature of the Witness Signature of the patient

Name:

Signature of Candidate **Signature of Supervisor**

मरीज अनुमति फार्म

तिथि _____

मैं _____ पत्नी/पुत्र/पुत्री _____

निवासी _____

इस शोधकार्य "**NON- DIABETIC KIDNEY DISEASES IN PATIENTS WITH DIABETES MELLITUS (CLINICO PATHOLOGICAL CORRELATION)"** में भाग लेने के लिए सहमत हूँ। मैं इस शोधकार्य में भाग लेने के लिए अपनी स्वेच्छा से तैयार हूँ। मैं पुष्टि करता/करती हूँ की यह स्वीकृति देने के लिए मुझे कोई आर्थिक प्रलोभन नहीं दिया गया और न ही दबाव डाला गया है। मुझे इस शोधकार्य का उद्देश्य स्पष्ट कर दिया गया है और मुझे यह विश्वास है की यह विज्ञान और मानवता के लाभ के लिए है। मुझे इस शोधकार्य में होने वाले जोखिम के बारे में समझा दिया गया है और मेरी समझ में यह जोखिम बहुत मामुली है।

मैं इस शोधकार्य में भाग लेने के सम्बन्ध में किसी भी समय अपनी अस्वीकृति व्यक्त कर सकता/सकती हूँ और मेरे अस्वीकृति व्यक्त करने का मेरे इलाज पर कोई प्रभाव नहीं पड़ेगा।

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गवाह के हस्ताक्षर रोगी के हस्ताक्षर/अंगूठे का निशान

नाम: नाम:

_____

अन्वेषक के हस्ताक्षर

_____

विद्यार्थी के हस्ताक्षर

ਮਰੀਜ਼ ਰਜ਼ਾਮੰਦੀ ਫਾਰਮ

ਮਿਤੀ_____

ਮੈ_____ਪਤਨੀ/ਪੁੱਤਰ/ਪੁੱਤਰੀ_____

ਵਾਸੀ _____

“NON- DIABETIC KIDNEY DISEASES IN PATIENTS WITH DIABETES MELLITUS (CLINICO PATHOLOGICAL CORRELATION)” ਤੇ ਚਲ ਰਹੀ ਖੋਜ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਲਈ ਰਜ਼ਾਮੰਦ ਹਾਂ। ਇਸ ਖੋਜ ਕੰਮ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਲਈ ਮੈ ਆਪਣੀ ਮਰਜ਼ੀ ਨਾਲ ਤਿਆਰ ਹਾਂ। ਮੈ ਇਹ ਦਸਣਾ ਚਾਹੁੰਦਾ ਹਾਂ/ ਚਾਹੁੰਦੀ ਹਾਂ ਕੇ ਇਹ ਰਜ਼ਾਮੰਦੀ ਦੇਣ ਵਾਸਤੇ ਮੈਨੂ ਕਿਸੇ ਵੀ ਤਰਾਂ ਦਾ ਲੋਭ-ਲਾਲਚ ਨਹੀ ਦਿੱਤਾ ਗਿਆ ਅਤੇ ਨਾ ਹੀ ਕੋਈ ਦਬਾ ਪਾਇਆ ਗਿਆ ਹੈ। ਮੈਨੂ ਇਸ ਖੋਜ ਦਾ ਮਨੋਰਥ ਸਮਝਾ ਦਿੱਤਾ ਗਿਆ ਹੈ ਅਤੇ ਮੈਨੂ ਇਹ ਭਰੋਸਾ ਦਵਾਇਆ ਗਿਆ ਹੈ ਕੇ ਇਹ ਖੋਜ ਵਿਗਿਆਨ ਅਤੇ ਮਾਨਵਤਾ ਦੀ ਭਲਾਈ ਲਈ ਹੈ। ਮੈਨੂ ਇਸ ਖੋਜ ਵਿਚ ਹੋਣ ਵਾਲੇ ਖਤਰੇ ਬਾਰੇ ਸਮਝਾ ਦਿੱਤਾ ਗਿਆ ਹੈ ਅਤੇ ਮੈਨੂ ਇਹ ਯਕੀਨ ਹੈ ਕੇ ਇਹ ਖਤਰਾ ਬਹੁਤ ਮਾਮੂਲੀ ਹੈ। ਮੈਨੂ ਇਸ ਖੋਜ ਕੰਮ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਦਾ ਢੰਗ – ਤਰੀਕਾ ਸਮਝਾ ਦਿੱਤਾ ਗਿਆ ਹੈ।

ਮੈ ਇਸ ਖੋਜ ਕੰਮ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਬਾਰੇ ਆਪਣੀ ਰਜ਼ਾਮੰਦੀ ਕਿਸੇ ਵੀ ਸਮੇ ਵਾਪਸ ਲੈ ਸਕਦਾ ਹਾਂ/ ਸਕਦੀ ਹਾਂ ਅਤੇ ਮੇਰੀ ਰਜ਼ਾਮੰਦੀ ਵਾਪਸ ਲੈਣ ਦਾ ਮੇਰੇ ਇਲਾਜ ਤੇ ਕੋਈ ਅਸਰ ਨਹੀ ਹੋਵੇਗਾ।

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ਗਵਾਹ ਦੇ ਹਸਤਾਖਰ ਮਰੀਜ਼ ਦੇ ਹਸਤਾਖਰ

ਮਿਤੀ_____ ਮਿਤੀ_____

ਪਤਾ _____ ਪਤਾ _____

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ਨਿਰੀਖਕ ਦੇ ਹਸਤਾਖਰ

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ਸਿਖਿਆਰਥੀ ਦੇ ਹਸਤਾਖਰ